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## The Incidence of Bovine Mastitis with Special Reference to the Non-specific Condition

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ALTHOUGH much information has been acquired in the past 30 years regarding the aetiology of bovine mastitis and considerable progress made in the treatment of the disease, the fact remains that it is still one of the most serious causes of loss to the dairy farmer. Some indication of the economic importance of mastitis was given in the Report of the Survey Committee of the National Veterinary Medical Association of Great Britain and Ireland, 1941. They estimated that of the 3,200,000 milk cows in Great Britain at that time, 800,000 or 25 per cent. had mastitis. Assuming that the average yield of milk per lactation for unaffected cows was 500 gallons, and the loss of milk due to mastitis was 60 gallons or 12 per cent., at 1s. 3d. per gallon this represented a monetary loss of £3,000,000 annually. The Committee also estimated that about 10 per cent. of the affected cows (80,000) had to be replaced each year because of mastitis at a cost of £2,000,000, *i.e.* £25 for each cow. Thus the total annual loss amounted to over £5,000,000. These are conservative figures and would now have to be increased considerably. The present average selling price of milk is about 3s. per gallon (Annual Review and Determination of Guarantees, 1955. H.M.S.O.) and recent work in this department indicates that the incidence of mastitis in this, the main dairy farming area in Scotland, is over 33 per cent. Further, in the United Kingdom in June, 1954, there were 3,730,000 cows yielding milk, this being the number of cows in calf at the June census (Commonwealth Economic Committee Intelligence Bulletin, February, 1955). Since the average annual yield per cow is now 670 gallons (Milk Marketing Board, 1954), a 12 per cent. loss in yield due to mastitis is equivalent to 80 gallons per cow. Thus the loss in milk yield can be estimated at about 100,000,000 gallons, which at present prices is approximately £15,000,000. With regard to losses due to stock replacement, about 124,000 cows have to be replaced each year because of mastitis (see above), and if the difference between the cost of the incoming cow and the outgoing cow is estimated conservatively at £30, the total cost of this item reaches £3,750,000; there is also an unknown sum representing the loss of income caused by a reduction in the number of surplus heifers for sale. The total annual loss therefore due to mastitis must now be about £19,000,000.

The dairy cow is particularly susceptible to mastitis no doubt because her mammary glands have been developed beyond natural requirements. As a result the udder has become what must be regarded as a pathologically hypertrophied organ, the size and pendulous character of which render it extremely liable to trauma. Moreover, the milking process when carried out carelessly, especially by machine, may, through mechanical injury, cause a chronic irritation of the udder tissues and predispose the quarter to infection (Meigs *et al.*, 1938; Watts, 1942; Little & Plastridge, 1946; Udall, 1947; Neave, Phillips & Mattick, 1952; Wilson, 1954). These factors reduce the average milking life of the dairy cow, and this, according to estimates by the Milk Marketing Board (1948) on the basis of records of 67,611 lactations, was only 3.1 lactations. The data of the Milk Marketing Board showed that animals which entered the milking herd aged two and a half years were as a rule discarded when they were only five and a half years old, which is about four or five years before a normal cow reaches maximum production.

The introduction of new methods of treatment by drugs and antibiotics raised hopes that the problem of mastitis would soon be solved, more especially since most of the cases were believed to be due to streptococci which are susceptible to antibiotic

therapy. However, the advisory work of this department, involving the examination of approximately 20,000 samples of milk yearly, indicates that the incidence of mastitis is unabated and this is confirmed by a study of the veterinary literature of this and other countries. To obtain more information on the point it was decided to compare our recent results with accepted data for the years prior to the introduction of the new methods of treatment.

**Methods of Diagnosis**

Mastitis in the incipient stage may be of such a mild nature that there are neither clinical symptoms in the animal nor obvious physical changes in the milk; accordingly laboratory methods are necessary for its diagnosis. These methods have as their object the detection of specific pathogens in the milk or increases in its cell content or changes in its biochemical or physical properties, such as electrical conductivity and chloride content. Most workers make use of culture tests as the chief method of diagnosis, but provided the cow is neither at the colostral nor the drying off stage, the total cell content of the milk is in our experience much the most reliable criterion of the disease (Malcolm, King & Campbell, 1942, 1944; Malcolm & Campbell, 1946; McFarlane, Blackburn, Malcolm & Wilson, 1949; Blackburn, Laing & Malcolm, 1955). and this has been the finding of other workers also (Cherrington, Hansen & Halversen, 1933; Holm, 1934; Hopkirk, 1935; Leitch, 1935; Hucker, Reed & Savage, 1937; Hucker, 1942; McEwan & Cooper, 1947; van Rensburg, 1947; Chu, 1949). Slanetz & Bartley (1953) did not consider that the " leucocyte " count was a reliable criterion of chronic mastitis (staphylococcal), but they based their diagnosis on a count of 1,000,000 per ml. or over, which in our experience is too high for diagnostic purposes. (With few exceptions no attempt has been made by the various workers, in carrying out cell counts of milk, to differentiate leucocytes from epithelial cells, even although the term " leucocyte count " has been used in recording results. Obviously the term leucocyte should not be used for an epithelial cell. It is worthy of note, however, that Blackburn *et al.* (1955) made differential counts of polymorphs, lymphocytes, and other cells (mostly epithelial) in a large number of milk samples, and found that in the diagnosis of mastitis the differential cell count showed no marked advantage over the total cell count, except in milk of late lactation.) Malcolm *et al.* (1942, 1944, 1946) and McFarlane *et al.* (1949) found further that mastitis organisms are frequently absent from the milk of quarters in which inflammation is present as indicated by a high cell content, and for this condition the term " non-specific " mastitis is used. Such non-specific forms of the disease have also been observed by Peterson, Hastings & Hadley, 1938; Peterson & Hastings, 1939; van Rensburg, 1947; Hughes, 1954. The presence of inflammation and the non-specific nature of the condition in such quarters have been confirmed by histological examination of the tissues by McFarlane *et al.* (1949), and also by the inability to isolate mastitis organisms from the tissues. Moreover, McFarlane *et al.* found that mastitis organisms are occasionally present in quarters which appear to be perfectly normal, *i.e.* manifesting no clinical symptoms of mastitis, the milk being normal both in its biochemical and physical properties and the cell content low. A number of these cases were examined *post mortem* and no histological lesions were found in the tissues, thus confirming that the quarters were free from mastitis. For these reasons, with cows in mid-lactation, the total cell content is used in our work as the criterion of the presence or absence of mastitis; and culture tests are employed to determine what specific pathogens, if any, are present.

The cell count was carried out throughout the whole of the work by the standardised method. This is a modification of the Breed's smear method, 0.01 ml. milk being measured by means of a standard platinum loop. The smears are fixed and stained by Newman's reagent, and the number of cells in 30 fields counted with 1/12 inch oil immersion objective. Where the cell content is high, over five cells per field, *i.e.* over 1,500,000 cells per ml. (the factor of 300,000 being used), there is no need to count more than 10 or 15 fields, provided the cells are evenly distributed. Under the low power of the microscope the uniformity of distribution can be readily observed to begin with and a rough indication of the cell count obtained. As a rule comparatively few organisms are observed in the direct smears even in the case of abnormal specimens.

Although it would appear from the results of our earlier work (see McFarlane *et al.*, 1949) that persistent cell counts of 100,000 per ml. indicate inflammatory changes in the udder, various points must be considered in interpreting the cell count. For instance, a high cell count from a particular quarter in the absence of mastitis organisms in cultures from the milk is not regarded as significant of mastitis unless it persists, because a *temporary* rise in cell content may be the result of mechanical injury. Further, factors other than mastitis may cause a considerable temporary rise in the cell content of the milk. For instance, prolonged retention of milk in the udder or some general infection of the animal may cause a rise in cell count, but in such cases the four quarters are affected simultaneously. Thus no particular figure is regarded as pathognomonic of mastitis with a random specimen of milk. We regard counts of over 250,000 per ml. in milk from an individual quarter as significant and over 500,000 per ml. as indicative of mastitis. Marked differences between the cell counts of the four quarters possess as much importance as the absolute figures, particularly when the counts are less than 500,000 per ml. Thus in a heifer, if the milk of one quarter persistently shows counts of 150,000 to 250,000 per ml., while the counts in the milk of the other three quarters are each less than 50,000, the condition is to be regarded as due to mastitis in the first quarter, even although mastitis organisms cannot be recovered from the milk. On the other hand, an older animal showing cell counts of 300,000 per ml. in all quarters with no organisms in the milk, is to be regarded as suspect but probably negative.

Herd tests are made by this department for farmers and veterinary surgeons where trouble has arisen owing to mastitis, the work being in part a continuation of the " Panel Scheme " of the National Veterinary Medical Association. However, by far the greater part of the work consists of herd tests carried out regularly for farmers as a precautionary measure. Thus our data can be looked on as representative of herd conditions in the West of Scotland. (Tests carried out on specimens of milk sent in from single cows suspected of having mastitis have not been included in the results.) In all these herd tests, mixed quarter samples from individual cows are examined for cell content, 200,000 cells per ml. being regarded as suspect. This figure or even 150,000 is preferable to the 250,000 diagnostic standard used for individual quarter samples, as it allows for the " diluting effect " of milk from any healthy quarters present. Obviously, where only one quarter is affected and the inflammation is of a mild character giving a cell count of about 500,000 for that quarter, it is possible that dilution with the normal milk from the other three quarters will reduce the count for the mixed sample to below 250,000. On the other hand, in our experience frequently where the mixed samples had cell counts of 200,000 per ml., subsequent examination of individual quarter samples showed the cows to be free from mastitis. This indicates that our diagnostic standard is sufficiently low to include all positive cows.

As a rule culture tests are not carried out on mixed quarter samples, but when such a sample yields a significant count, separate quarter samples from the cow are tested immediately both by cell count and culture. In the latter, a 0.01 ml. loopful of milk from each quarter is spread on blood agar and the various types of colonies which develop are examined and subcultures made. Whenever possible, streptococci are grouped according to the methods of Lancefield and Minett, and staphylococci are typed according to their action on blood and pigment production.

## Results

I. *Incidence of Mastitis, Specific and Non-Specific*

The results of herd tests for the four years 1951-54 are given in Table I and Fig. 1A. Cases of mastitis are grouped according to the types of mastitis organisms found in the milk; those definitely positive by the cell count test but which yielded no mastitis organisms on culture are designated " non-specific mastitis." It is seen that about 4,000 cows were examined each year and that the proportion of affected cows varied from 34 to 41 per cent. (average 37 per cent.). Of all the animals examined, 23 per cent. had specific mastitis and 14 per cent. non-specific, and there was no significant variation in these proportions from year to year.

Comparison with our data for earlier years is interesting. Table III gives the incidence of mastitis in 2,303 cows tested during the two years, 1947-49; the average incidence was 35 per cent. In this case 25 per cent. of the cows examined had specific mastitis and 10 per cent. non-specific. In another series of tests carried out in 1941 on 1,098 cows, 44 per cent. showed evidence of mastitis.

Data for years prior to 1941 are also available from other sources. In the Report of the Survey Committee of the National Veterinary Medical Association of Great Britain and Ireland, 1941, it was estimated that 25 per cent. of the milk cows in Great Britain and Ireland had mastitis. Stableforth (1938) stated that the incidence of mastitis varied from 10 to 80 per cent. in different herds, the average being about 30 per cent. In the Report of the Committee on Cattle Diseases, 1934, it was concluded that 30 per cent. of the milking cattle in this country were affected with mastitis other than the tuberculous form. This Report also quotes the results of an investigation carried out just previous to 1934 by the Research Institute of Animal Pathology of the Royal Veterinary College, London; 1,613 cows from 30 herds were examined and " 660 (40.9 per cent.) gave samples of milk having definite infection of the udder, usually of a latent character." This was regarded as rather a high incidence, but the report mentioned that most of the herds were examined because there had been trouble in them with mastitis.

Thus, according to various authorities, it would appear that prior to about 1941 the average incidence of mastitis in dairy cows in the United Kingdom was from 25 to 30 per cent. This figure, however, is 10 to 15 per cent. lower than those obtained by us, and the question arises as to whether there has been an actual increase in the incidence of mastitis in the past 15 years or whether our higher figures are due to the fact that, unlike most other workers, we use the cell count test in addition to the culture test in the diagnosis of the disease, so that subclinical cases of non-specific mastitis are included.

II. *Relative Incidence of Non-specific Mastitis and the Different Types of Specific Mastitis*

(a) *Data based on all cows examined.*—Table I and Fig. 1A show that in the period 1951-54 about 12 per cent. of the cows examined had staphylococcal mastitis, 6 per cent. streptococcal, 5 per cent. a mixed staphylococcal and streptococcal infection and 14 per cent. non-specific mastitis. Table III gives the data for the period 1947-49, but in this case mixed staphylococcal and streptococcal infections were included amongst streptococcal cases, except in a few instances where staphylococci appeared to be predominant. However, in view of the proved importance of staphylococci as pathogens and their appearance as pure infections, there is no justification for assuming that in cases where mastitis streptococci occur along with staphylococci, the latter are devoid of pathogenic importance. Allowing for this difference in recording the results of the mixed infections, a comparison of the data for 1951-54 with those for 1947-49 shows only a slight increase in the incidence of staphylococcal infections (about 2 per cent.) in the later period; a reduction of 4 per cent. in the streptococcal cases (including mixed infections) and an increase of 4 per cent. in the non-specific. Our figures for periods before 1947 are not strictly comparable with the above, since prior to 1947 selective culture media were largely used, which permitted the growth of streptococci while inhibiting staphylo-

TABLE I

INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 TO 1954 INCLUSIVE

| Year | Total cows | Cows with mastitis * | | Proportion of *all* cows (affected and unaffected) with the various types of mastitis | | | |
|---|---|---|---|---|---|---|---|
| | | | % | Staphylococcal % | Streptococcal % | Staphylococcal and Streptococcal % | Non-specific % |
| 1951 | 4,293 | 1,528 | 35·6 | 12·2 | 5·3 | 4·0 | 13·7 |
| 1952 | 4,762 | 1,800 | 37·8 | 13·1 | 6·0 | 5·1 | 13·4 |
| 1953 | 4,205 | 1,705 | 40·6 | 12·5 | 7·0 | 5·4 | 15·3 |
| 1954 | 3,656 | 1,235 | 33·8 | 10·7 | 5·3 | 3·4 | 14·4 |
| 1951–54 | 16,916 | 6,268 | 37·1 | 12·2 | 5·9 | 4·6 | 14·2 |

* 21 cows had coliform mastitis ; 1 cow had summer mastitis due to *Corynebacterium pyogenes*.

TABLE II

INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 TO 1954 INCLUSIVE

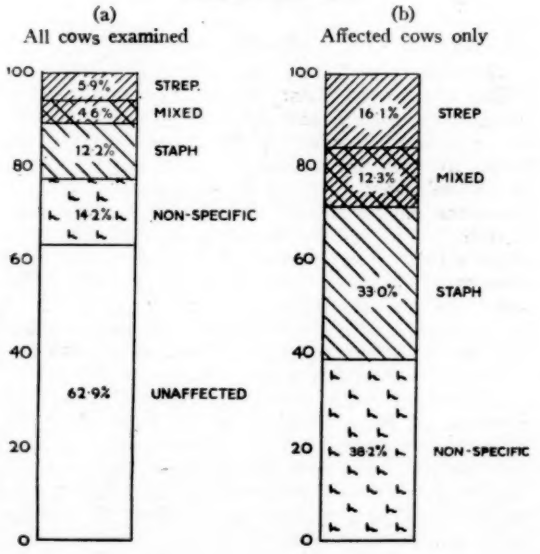
| Year | Cows with mastitis | Proportion of *affected* cows with the various types of mastitis | | | |
|---|---|---|---|---|---|
| | | Staphylococcal % | Streptococcal % | Staphylococcal and Streptococcal % | Non-specific % |
| 1951 | 1,528 | 34·2 | 15·0 | 11·2 | 38·7 |
| 1952 | 1,800 | 34·7 | 16·0 | 13·5 | 35·5 |
| 1953 | 1,705 | 30·9 | 17·3 | 13·5 | 37·8 |
| 1954 | 1,235 | 31·8 | 15·7 | 10·1 | 42·2 |
| 1951–54 | 6,268 | 33·0 | 16·1 | 12·3 | 38·2 |

cocci. However, figures are available from other sources; for instance, in the Report of the Committee on Cattle Diseases, 1934 (see above), it was stated that 90 per cent. of the affected animals had streptococcal mastitis. This would give 27 per cent. as an estimate of the incidence of streptococcal mastitis in the country in 1934, *i.e.* the time of the report, or 22 per cent. in 1941, at the time of the Report of the Survey Committee of the National Veterinary Medical Association of Great Britain and Ireland, 1941 (see above). Further, it was agreed that 85 to 95 per cent. of the streptococcal infections were caused by *Str. agalactiae* (see also Stableforth, 1938). With regard to the incidence of staphylococcal mastitis there is considerable variation in the findings of different workers, no doubt partly due to the fact that the figures in some instances are based on the results of a relatively small number of herds. (Hopkirk (1935), 30 per cent.; Gwatkin, Hadwen & Legard (1936), 21 per cent.; Plastridge, Weirether and Williams (1937), 10 to 35 per cent.; Stableforth (1938), " relatively uncommon "; Miller (1943), 33.3 per cent.; Cunningham *et al.* (1947), 10 per cent.; Hughes (1954), 20.3 per cent.) Later, Stableforth (1953) and Hughes (1954) found that staphylococci were responsible for 18 per cent. and 12.6 per cent. respectively of cases of clinical mastitis. As a result of this wide variation in the data it is difficult to estimate the average figure for the incidence of staphylococcal mastitis in the United Kingdom. However, if the conclusion of the Committee on Cattle Diseases (1934) is accepted that 90 per cent. of the

FIG. 1.

INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 - 1954

(TABLES 1 AND 2)



cases of mastitis were caused by streptococci, then not more than 10 per cent. of the cases could have been caused by staphylococci or less than 3 per cent. of all the milk cows. According to our results the incidence of staphylococcal mastitis amounts to about 12 per cent. of all milk cows, and the figures for 1947-49 agree closely with those for 1951-54. With regard to the incidence of non-specific mastitis, comparatively little information is available from other sources. Until recently only a few workers recognised the condition, especially when subclinical (Peterson *et al.*, 1938, 1939; Malcolm *et al.*, 1942, 1944, 1946; van Rensburg, 1947; McFarlane *et al.*, 1949). However, from our results in herd tests about 15 years ago (Malcolm *et al.*, 1942, 1944), it appears that even then such cases were frequent, and Tables III and I show that in 1947-49, 10 per cent. of the animals examined had non-specific mastitis, and in 1951-54, 14 per cent.

(b) *Data based on affected cows.*—A clearer indication of the relative incidence of the different types of mastitis can be obtained by comparing their proportions among *mastitis-affected* cows. Thus in 1951-54 (see Table II and Fig. 1B), 38 per cent. of affected cows had the non-specific form of the disease, 33 per cent. had staphylococcal mastitis, and 28 per cent. had streptococcal or a mixed streptococcal and staphylococcal infection (only 16 per cent. being pure streptococcal infections). In the period 1947-49 (see Table IV), 28 per cent. of affected cows had the non-specific form of the disease, 28 per cent. had staphylococcal mastitis and 44 per cent. had streptococcal or a mixed streptococcal and staphylococcal infection. Accordingly, there has been a considerable increase in the incidence of non-specific mastitis since 1947; a slight increase in the purely staphylococcal infections; and a definite fall in the streptococcal or mixed streptococcal and staphylococcal infections. Consequently, instead of the bulk of mastitis cases being, as formerly, due to streptococci (in the opinion of some workers, 85 to 95 per cent. of the cases), the present work shows that streptococcal infections are far outnumbered by staphylococcal and non-specific cases respectively.

(c) *Data based on all quarters examined.*—As previously stated, in the examination of herds a mixed quarter sample of milk from each cow was first tested for cell content and on this basis definitely negative cows were eliminated, *i.e.* those with cell counts of less than 200,000 per ml. Individual quarter samples of milk from all the other cows were then examined, both the cell count and culture tests being employed. Table V and Fig. 2A give the incidence of the different types of mastitis, expressed as proportions of the total number of quarters examined. In order to provide an accurate estimate of the incidence of the disease in individual quarters, cows which were found to be negative in the mixed quarter sampling of the herd test have been included in the table, being represented in each case as four negative quarters. Thus the table gives data for 67,664 quarters. It will be seen that an average of about 17,000 quarters was examined each year and that the proportion of affected quarters varied from 21 to 25 per cent., the average for the four years being 23 per cent. There was no significant variation from year to year either in the proportion of affected quarters or of quarters affected with the different types of the disease. Of the total quarters (affected and unaffected) examined during the four years, 5.7 per cent. had staphylococcal mastitis, 3.4 per cent. had streptococcal, 0.6 per cent. had a mixed staphylococcal and streptococcal infection and 12.4 per cent. had non-specific mastitis.

TABLE III

INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS IN 1947 TO 1949

| Year | Total cows | Cows with mastitis | % | Proportion of *all* cows (affected and unaffected) with the various types of mastitis: Staphylococcal % | Streptococcal and mixed infection % | Non-specific % |
|---|---|---|---|---|---|---|
| 1947–48 | 1,090 | 428 | 39·3 | 9·7 | 18·0 | 11·4 |
| 1948–49 | 1,213 | 385 | 31·7 | 10·3 | 12·7 | 8·6 |
| 1947–49 | 2,303 | 813 | 35·3 | 10·0 | 15·3 | 9·9 |

TABLE IV

INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS IN 1947 TO 1949

| Year | Total cows | Cows with mastitis | Proportion of *affected* cows with the various types of mastitis: Staphylococcal % | Streptococcal and mixed infection % | Non-specific % |
|---|---|---|---|---|---|
| 1947–48 | 1,090 | 428 | 24·7 | 46·2 | 28·9 |
| 1948–49 | 1,213 | 385 | 32·4 | 40·5 | 27·0 |
| 1947–49 | 2,303 | 813 | 28·4 | 43·5 | 28·0 |

(d) *Data based on affected quarters.*—Table VI and Fig. 2B give the proportion of the different types of mastitis calculated on the number of affected quarters and not on the total number of quarters. The table and figure show that the staphylococcal and non-specific forms of the disease each far outnumbered those due to streptococci. The non-specific form constituted 54 per cent. of the mastitis-affected quarters; the staphylococcal form 25 per cent.; and the streptococcal only 15 per cent. Only 2.7 per cent. of the affected quarters had a mixed infection of streptococci and staphylococci. The last figure is much lower than that for the mixed infection shown for cows (see Table II and Fig. 1B), since the streptococci and staphylococci usually occurred in different quarters.

## Discussion and Conclusions

In spite of the new methods of treatment by chemotherapeutic drugs and antibiotics, mastitis continues to be a major problem for the dairy industry. In the advisory work of this department, up to 20,000 samples of milk are still being examined for the disease each year. During the four years 1951-54 the results of 67,664 samples of milk from the individual quarters of cows which had not been recently treated for the disease, showed that 34 to 41 per cent. of the animals had mastitis. These figures agree fairly closely with those for earlier periods, 1947-49 and also 1941, but are about 10 to 15 per

FIG. 2.
INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 - 1954
(TABLES 5 AND 6)

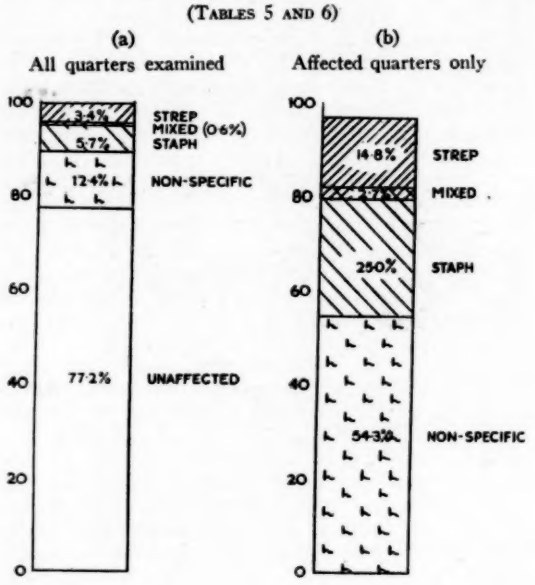



TABLE V
INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 TO 1954 INCLUSIVE
(expressed in terms of individual quarters)

| Year | Total quarters | Affected quarters * | % | Proportion of *all* quarters (affected and unaffected) with various types of mastitis: Staphylococcal % | Streptococcal % | Staphylococcal and Streptococcal % | Non-specific % |
|---|---|---|---|---|---|---|---|
| 1951 | 17,172 | 3,788 | 22·1 | 5·6 | 2·9 | 0·5 | 12·3 |
| 1952 | 19,048 | 4,502 | 23·6 | 6·3 | 3·4 | 0·8 | 12·4 |
| 1953 | 16,820 | 4,148 | 24·7 | 6·0 | 4·1 | 0·7 | 13·1 |
| 1954 | 14,624 | 3,017 | 20·6 | 4·7 | 3·0 | 0·5 | 11·8 |
| 1951–54 | 67,664 | 15,455 | 22·8 | 5·7 | 3·4 | 0·6 | 12·4 |

* 470 blind quarters have been included in the affected quarters, as this condition is most commonly due to mastitis ; 28 of the positive quarters were infected with coliform bacteria and one with *Corynebacterium pyogenes*.

TABLE VI
INCIDENCE OF BOVINE MASTITIS IN DAIRY HERDS FROM 1951 TO 1954 INCLUSIVE
(expressed in terms of individual quarters)

| Year | Affected quarters | Proportion of *affected* quarters with the various types of mastitis: Staphylococcal % | Streptococcal % | Staphylococcal and Streptococcal % | Non-specific % |
|---|---|---|---|---|---|
| 1951 | 3,788 | 25·6 | 13·3 | 2·2 | 55·5 |
| 1952 | 4,502 | 26·7 | 14·5 | 3·2 | 52·4 |
| 1953 | 4,148 | 24·4 | 16·5 | 2·7 | 53·0 |
| 1954 | 3,017 | 22·6 | 14·6 | 2·2 | 57·3 |
| 1951–54 | 15,455 | 25·0 | 14·8 | 2·7 | 54·3 |

cent. higher than the estimates of 25 to 30 per cent. made for the United Kingdom by various authorities for years prior to 1941 (Report of Committee on Cattle Diseases, 1934; Stableforth, 1938; Report of the Survey Committee of the National Veterinary Medical Association of Great Britain and Ireland, 1941). Our higher figures are not to be regarded as indicating a subsequent increase, but rather as due to the use of the cell count test in addition to culture tests in the diagnosis of the disease. In consequence, cases of non-specific mastitis are included, amounting to about 14 per cent. of all cows examined; these if subclinical could not be detected by culture tests alone. Therefore it appears that there has been no significant change in the incidence of mastitis in this area since 1941.

It must be emphasised that *non-specific mastitis* diagnosed on the basis of an increased cell count of the milk without demonstrable bacteria was almost invariably associated with histological changes of an inflammatory nature in the udder tissues, and in most of these cases no mastitis organisms were obtained in cultures from the lesions. This applied even to early subclinical mastitis in heifers (Malcolm *et al.*, 1942; McFarlane *et al.*, 1949). Moreover, the converse was found to be true, *i.e.* acute inflammatory lesions in the udder, with the exception of minute localised lesions, were always associated with a high cell count of the milk (Blackburn, 1955). McFarlane (1948) examined a series of udders (from all types of cases, specific and non-specific) by histological methods as well as bacteriologically and found in 175 pathologically positive quarters from heifers and older cows that no mastitis organisms could be obtained from the tissues of 59 (34 per cent.). No pathogens could be found in the tissues of 23 per cent. of 92 of them which showed " diffuse progressive mastitis often of severe or moderately severe " grade, or from 39 per cent. of 39 which showed " diffuse dormant mastitis " and 52 per cent. of 44 which showed " progressive and focal dormant mastitis." Such non-specific conditions cannot be ignored, since they may cause a reduction in the yield of milk with lowering in the quality, especially in solids-not-fat (Waite & Blackburn, 1955), and may lead to permanent damage and even progress to atrophy of the quarter. It may be objected that conditions affecting the general health of the cow or functioning of the udder, *e.g.* premature drying-off, faulty machine-milking, etc., can cause an increase in the cells in the milk; but such conditions are likely to affect all the quarters equally and not, as in the above cases, isolated quarters. Again, cases of non-specific mastitis have been ascribed to so-called " physiological breakdown " of the mammary tissues; but if there is such a condition, it would be liable to affect more than one quarter (general distribution) and it would not commonly develop in heifers in first lactation, whereas non-specific mastitis of limited distribution occurs frequently in young animals (Malcolm *et al.*, 1944).

It might be objected that so-called non-specific mastitis is merely a temporary manifestation of trauma. In opposition to this it must be emphasised that the condition referred to here can persist throughout a whole lactation and even be present in a subsequent lactation. Also, it may be progressive, as shown by MacFarlane, see above, and Blackburn (1955), and eventually lead to atrophy of the whole quarter. Whatever may be the initial exciting factor, it is clear we have to deal with a progressive disease which eventually destroys the physiological function of the mammary tissue.

The question arises as to the aetiology of non-specific mastitis; there are two chief possibilities:— (1) There may have been a definite bacterial infection initially, the organisms subsequently having disappeared from the lesions. According to Petersen (1955) the persistence of the non-specific condition may be due to the development of allergy to the organisms by the mammary tissues. The fact that organisms could not be found in the milk even at an early stage may have been due to their being retained within the tissues and so not reaching the milk ducts and the milk. (2) The disease may be initiated by some non-bacterial factor, *e.g.* trauma, and later streptococci, staphylococci, or coliform organisms may be superadded and appear in the milk. These organisms would then be secondary invaders, commensals or contaminants, which were enabled to obtain a foothold owing to the prior abnormal condition. The claim by some workers that organisms can almost always be obtained in commencing cases of mastitis may rest on the use only of clinical and culture tests and not the cell count, so that subclinical mastitis is not detected at an early enough stage. Where an animal has a staphylococcal or streptococcal infection in one quarter and non-specific mastitis in another, it may be argued that the non-specific condition in the second quarter is merely a concomitant reaction to the specific infection in the first. If this were so, it would be expected that the non-specific reaction would occur to some extent in all the other quarters. However, this was not the case, since on taking into account all cows with staphylococcal mastitis, the ratio of the total of infected to non-specific quarters was 2.1:1; similarly with streptococcal mastitis, the ratio was 2.5:1. Thus at least one quarter was unaffected as a rule. Again, if the non-specific condition was associated directly with a specific infection in another quarter, the frequency of the non-specific condition would most probably vary according to the nature of the infecting agent; but the incidence of non-specific mastitis was about the same irrespective of whether the infection in another part of the udder was staphylococcal or streptococcal. Further, it must be remembered that non-specific mastitis occurs mainly in absence of a specific infection in any other quarter; thus in about 62 per cent. of our series the non-specific quarters were from cows not affected with specific mastitis.

In addition to the naturally occurring cases of non-specific mastitis discussed above, others arise from specific infections, especially streptococcal, which have been treated with drugs or antibiotics. Such treatment may cause the organisms to disappear from the milk while the cell count remains high as an indication that the inflammatory condition persists. Stableforth (1953) obtained no mastitis organisms in

the milk of 27 per cent. of 2,005 cases of clinical mastitis examined in 1952 and suggested that this high figure was due to treatment of the cows prior to sampling. However, this could not have been the cause of the occurrence of purely non-specific forms of mastitis in the first lactation heifers examined by us in 1942-44, as these had never been treated with drugs or antibiotics (Malcolm *et al.*, 1944). Also, it would not apply to Hughes' (1954) finding that over the five years 1949-53, 18.8 per cent. of 2,684 clinical cases of mastitis were non-specific, since the animals were examined prior to treatment. This proportion would be higher if some of his 5.5 per cent. " contaminated " samples contained no mastitis organisms. The cows from which our data have been obtained had not been treated just prior to testing, although it is possible that they may have been treated some weeks or months earlier.

With regard to *the incidence of the various types of mastitis*, it was commonly accepted, as previously mentioned, that up to the introduction of treatment on a large scale by antibiotics in 1947, 22 to 27 per cent. of all milking cows had streptococcal mastitis. We found that in the period 1951-54 only 6 per cent. of the total number of cows examined had streptococcal infections or, including the mixed streptococcal and staphylococcal cases, 11 per cent. In 1947-49 about 15 per cent. had streptococcal or mixed streptococcal and staphylococcal infections. There has therefore been a considerable reduction in the incidence of streptococcal mastitis since 1941 and even in the last few years. With regard to staphylococcal cases, from the data available from other sources, it would appear that prior to 1947 only 3 per cent. of all milking cows had this form of the disease. However, in herd tests carried out by us before 1947, staphylococcal mastitis was found to be frequent; and our latest results show that the incidence of this form of mastitis is still considerable, amounting to about 12 per cent. of all the cows examined or about 33 per cent. of all cases of mastitis, specific and non-specific. The data are remarkably uniform over the two periods 1951-54 and 1947-49 and, in view of the large number of animals examined, may be taken as a reliable indication of the incidence of staphylococcal mastitis in the West of Scotland. Our results for 1951-54 show that 14 per cent. of the cows had non-specific mastitis, equivalent to 38 per cent. of all mastitis-affected cows, while in the period 1947-49 the corresponding figures were 10 per cent. and 28 per cent. respectively. The findings of Hughes (1954) and Stableforth (1953) that 18.8 per cent. and 27 per cent. respectively of cases of *clinical* mastitis were non-specific are much below our figure; this is no doubt due to our use of the cell count as well as the culture test in the diagnosis of the disease.

Whereas prior to 1941 the bulk of cases were due to streptococci, these are now far outnumbered by either non-specific or staphylococcal forms, *viz.*, 38 per cent. non-specific, 33 per cent. staphylococcal, 16 per cent. streptococcal, and 12 per cent. mixed streptococcal and staphylococcal infections. The considerable reduction in the incidence of streptococcal mastitis may be due to the fact that it can be successfully treated, but there has been no reduction in staphylococcal mastitis due to treatment as the staphylococci frequently become resistant to penicillin and other chemotherapeutic agents. The high proportion of non-specific mastitis is accounted for by the fact that, as already mentioned, antibiotic treatment of specific mastitis, especially streptococcal, may result in no pathogens being found later in the milk, although the cell content may remain high.

A more accurate estimation of the incidence of bovine mastitis can be made if the data from individual quarters are considered instead of from individual cows, since each quarter should be regarded as a separate and independent unit, a fact which is accepted by veterinary practitioners and research workers in general. Thus in our experience a cow may have a *Str. agalactiae* infection in one quarter, a staphylococcal in a second quarter, and perhaps a *Str. dysgalactiae* or a non-specific mastitis in a third. These conditions may persist unchanged in the different quarters over one or more lactations. Infection of one quarter from another may occur through the teat orifices or wounds or sores, and also perhaps through the blood stream, but there is little likelihood of organisms passing directly from one quarter to another through the intervening tissues (Blackburn, 1955). This is shown by the fact that although 12.3 per cent. of all *mastitis-affected cows* (see Table II) were infected with two types of organisms, only 2.7 per cent. of all *affected quarters* had a mixed infection (see Table VI). Even an infection so acute as *Corynebacterium pyogenes* mastitis (" summer mastitis ") tends to remain localised in one or two quarters.

According to the data from 67,664 quarter specimens, non-specific mastitis constituted about 54 per cent. of the affected quarters, staphylococcal 25 per cent., and purely streptococcal infections only 15 per cent. The fact that over half of the affected quarters were non-specific is worthy of note, because even at the present time this form of mastitis, unless clinical, is largely unrecognised or ignored in spite of its effect on the composition of the milk and on the milking life of the cow. It is difficult to understand how a persistently high cell content, *e.g.* 1,000,000 per ml., in the milk of one or more quarters, can be regarded as of no significance on the ground that because no organisms are found in the milk, the condition is not mastitis. The fact remains that if the tissues of such abnormal quarters are examined histologically, inflammatory changes can as a rule be found (McFarlane *et al.*, 1949). Moreover, the importance of staphylococcal mastitis does not appear to have been appreciated by many workers in the past, although this condition is responsible for 25 per cent. of the mastitis-affected quarters in the West of Scotland. It is frequently claimed that mastitis organisms are harmless to man. This may apply to certain streptococci, *e.g.*, *Str. agalactiae*, but it is questionable whether the same can be said about haemolytic staphylococci, or coliform organisms associated with coliform mastitis.

**Summary**

(1) Bovine mastitis is still a formidable economic problem in spite of modern chemotherapy and antibiotics.

(2) The diagnosis depends upon laboratory tests of the milk as well as clinical signs. Among the former, the cell count is of very great importance, as it may be the only positive test in early progressive cases of the disease.

(3) In the West of Scotland the average incidence of mastitis in recent years is 37 per cent. of all cows. In about three-fifths of the cases specific pathogenic organisms are present in the milk (specific mastitis). But in the remaining two-fifths, organisms are not demonstrable either in the milk or udder tissues (non-specific mastitis). It is stressed that in the investigation of mastitis the unit is the quarter and not the cow.

(4) Of the *specific* infections, about three-quarters are now due to staphylococci (in small proportion mixed staphylococcal and streptococcal infections), the rest are chiefly streptococcal; these last alone are highly responsive to chemotherapy or antibiotics.

(5) Non-specific mastitis has been largely neglected in the past, yet its practical importance is great. Its occurrence dates from before the era of chemotherapy and antibiotics. Its nature is discussed.

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**References**

Annual Review and Determination of Guarantees, 1955. H.M.S.O.

BLACKBURN, P. S. (1955). Private communication.

———, LAING, C. M., & Malcolm, J. F. (1955). *J. Dairy Res.* **22.** 37.

CHERRINGTON, V. A., HANSEN, H. C., & HALVERSEN, W.V. (1933). *J. Dairy Sci.* **16.** 59.

CHU, S. J. (1949). *J. comp. Path.* **59.** 81.

Commonwealth Economic Committee. Intelligence Bulletin, Feb. 1955.

CUNNINGHAM, A., WILSON, M. A., INESON, P. J., RAYMONT, J. K. B., & Mannan, K. A. (1947). *Tech. Bull.* No. 2, Sept., 1947. Edinburgh and East of Scotland College of Agriculture.

Economic Advisory Council, *Rpt. Comm. Cattle Dis.* (1934). 161.

GWATKIN, R., HADWEN, S., & LEGARD, H. M. (1936). *Canad. publ. Hlth. J.* **27.** 391.

HOLM, G. C. (1934). *J. Amer. vet. med. Ass.* **85.** 735.

HOPKIRK, C. S. M. (1935). *Res. Rep. N. Z. Dep. Agric.*

HUCKER, G. J. (1942). *J. Milk Tech.* **5.** 323.

———, REED, W. S., & SAVAGE, E. S. (1937). *Tech. Bull. N.Y. St. agric. Exp. Sta.* No. 244.

HUGHES, D. L. (1954). *Vet. Rec.* **66.** 17.

LEITCH, R. H. (1935). *Bull. No.* 130 *West of Scotland Agric. Coll.*

LITTLE, R. B., & PLASTRIDGE, W. N. (1946). "Bovine Mastitis" 402 and 469. McGraw-Hill Book Company Inc., New York.

MCEWAN, A. D., & COOPER, M. B. (1947). *Vet. Rec.* **59.** 655.

MCFARLANE, D. (1948). Unpublished.

———, BLACKBURN, P. S., MALCOLM, J. F., & WILSON, A. L. (1949). *Vet. Rec.* **61.** 807

MALCOLM, J. F., KING, C. W., & CAMPBELL, M. M. (1942). *Proc. Soc. agric. Bact.* 30.

———, ———, & ———. (1944). *Vet. Rec.* **56.** 227.

———, & CAMPBELL, M. M. (1946). *Proc. Soc. agric. Bact.* **1.** 29.

MEIGS, E. B., CONVERSE, H. T., BURKEY, L. A., ROGOSA, M., & SANDERS, G. P. (1938). *J. Dairy Sci.* **21.** 165–167. (abstract).

Milk Facts and Figures. Milk Marketing Board, No. 9, April, 1948. Dairy Husbandry 111. The Bureau of Records.

Milk Marketing Board. Report of the Production Division. No. 5, 1954.

MILLER, W. T. (1943). *J. Bact.* **45.** 307.

National Veterinary Medical Association of Great Britain and Ireland. Report on Diseases of Farm Livestock, 1941. *Vet. Rec.* **53.** 3.

NEAVE, F. K., PHILLIPS, M., & MATTICK, A. T. R. (1952). *J. Dairy Res.* **19.** 14.

PETERSEN, W. E. (1955). *Aust. J. Dy. Tech.* **10.** 117.

PETERSON, E. H., & HASTINGS, E. G. (1939). *Cornell Vet.* **29.** 11.

———, ———, & HADLEY, F. B. (1938). *Ibid.* **28.** 307.

PLASTRIDGE, W. N., WEIRETHER, F. J., & WILLIAMS, L. F. (1937). *J. Bact.* **35.** 66.

RENSBURG, S. W. J. van. (1947). *Onderstepoort J. vet. Sci.* **22.** (1).

SLANETZ, L. W., & BARTLEY, C. H. (1953). *J. infect. Dis.* **92.** 139.

STABLEFORTH, A. W. (1938). *Agricultural Progress.* **15.** 8.

———. (1953). *Vet. Rec.* **65.** 709.

UDALL, D. H. (1947). *Cornell Vet.* **37.** 73.

WAITE, R., & BLACKBURN, P. S. (1955). Private communication.

WATTS, P. S. (1942). *Vet. Rec.* **54.** 181.

WILSON, C. D. (1954). British Veterinary Association, Annual Congress, Sept., 1954.

## WORLD PRODUCTION OF MILK AND EGGS

World milk production continues to move steadily upwards according to the Food and Agriculture Organisation. The figures show an increase of 14 per cent. over those for the years immediately before the Second World War. By far the greatest part of 1954 milk production—225.2 million tons—came from cows. Buffalo produced 15.5 million tons, goats 7.7 million tons, and sheep 4.8 million tons. Practically the whole of the increase in world milk production since before the war has been in cows' milk. There has been virtually no change in production of other types of milk.

The highest reported yields per milking cow in 1954 were for the Netherlands. High yields were also reported from Belgium, Israel, Denmark, Luxemburg, and Hawaii. United Kingdom yields came after these.

World hen-egg production (excluding the U.S.S.R.) reached 10.25 million tons in 1954. The 1934-38 annual average was 7.01 million tons, so the increase over pre-war production was approximately 46 per cent. The greatest production increase was from the North American region—an increase of approximately 79 per cent. European production increased about 26 per cent

# Exudative Pleurisy in the Cat

BY

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**Norwich**

IN the past, the writer often encountered cases of exudative pleurisy in adult cats, which on *post-mortem* examination revealed a picture resembling that described for pulmonary tuberculosis in this species. Samples of exudate sent for bacteriological examination invariably produced a report that they were bacteriologically sterile. It was therefore decided to investigate this particular condition with a view to elucidating its aetiology and actual pathology. Accordingly, samples of pleural exudate were obtained aseptically from further cases and, together with samples of lung tissue, were despatched to the laboratory.

The following cases may help to establish the clinical and *post-mortem* picture of this condition.

### Case 1

*Subject.*—Three-year-old neuter cat.

This animal had been ill about one week before advice was sought. There was complete anorexia, the cat was very listless and the respirations were laboured with some degree of mouth breathing. Temperature was 101.5° F. Auscultation showed very muffled heart and lung sounds, and percussion revealed general dullness over both sides of the thorax. Destruction was advised and the whole carcase was forwarded to the laboratory.

*Post-mortem.*—The chest cavity was almost full of an offensive brown fluid, the lungs partially collapsed and covered with greyish deposits of fibrin.

*Bacteriology.*—Cultures of the exudate proved to be sterile.

*Histology.*—Examination of the lungs showed the presence of massive numbers of the larvae of *Aleurostrongylus abstrusus.*

### Case 2

*Subject.*—Six-year-old neuter cat.

The cat had been ill for a week with complete anorexia for the last three days before advice was sought. The animal was very listless and preferred to lie on its sternum with forepaws extended. The respirations were somewhat laboured and the heart sounds muffled, although the lung sounds were fairly clear. Percussion of the thorax revealed dullness on both sides of the chest cavity. Body temperature was 102.5° F. Penicillin and triple sulphonamide association* were prescribed.

Three days later, the cat's condition had deteriorated; respirations were now dyspnoeic and there was continuous mouth breathing. Both lung and heart sounds were now muffled, and both sides of the thorax were completely dull to percussion. The pulse was thin and thready.

The animal was destroyed, and several c.c. of pleural exudate were aspirated aseptically and, with a portion of lung tissue, despatched to the laboratory.

*Post-mortem.*—The chest cavity was filled with an opaque fluid with shreds of fibrin floating in it. The lungs were collapsed and with the pericardium were partially covered with greyish fibrinous deposits. Some adhesions between the lungs and the chest wall were noted.

*Bacteriology.*—Cultures of the exudate proved to be sterile.

*Histology.*—A considerable number of the larvae of *Aleurostrongylus abstrusus* were again present.

### Case 3

*Subject.*—Four-year-old neuter cat.

The cat had been ill about five days, but had been taking food up to the day previous to being presented for examination; it was very lethargic, and cried when picked up or when any pressure was applied to the chest. When left alone the cat tended to lie on its sternum with its head on its extended forepaws. Respirations were very laboured, especially on inspiration. Body temperature was 102° F. Auscultation revealed muffled heart sounds and no lung sounds on the left side of the thorax, but more pronounced heart sounds and muffled lung sounds on the right side of the thorax. Percussion gave pronounced dullness on the left side and diminished resonance on the right side of the chest.

The animal was destroyed, and fluid was aspirated aseptically for bacteriological examination.

*Post-mortem.*—The chest cavity was filled with a yellowish opaque fluid with a very offensive smell and clots of fibrin suspended in it. The pericardium and lungs were covered with a greyish deposit and there were many adhesions between the pleural surfaces.

*Bacteriology.*—A haemolytic streptococcus was isolated from the exudate.

*Histology.*—Typical lesions of an extensive broncho-pneumonia present.

### Case 4

*Subject.*—Six-year-old neuter cat.

This animal had been ill for about three days. Respirations were gasping in character and made through an open mouth; the cat cried when any pressure was applied to the chest. When left to its own devices the animal always resumed the prone position on its sternum with its forelegs and head extended. Body temperature was 102° F. Percussion revealed pronounced dullness on both sides of the thorax and the heart and lung sounds were muffled on auscultation. Penicillin was administered but the cat died later the same day.

*Post-mortem.*—The chest cavity was filled with a reddish opaque fluid with a very offensive smell and clots of fibrin floating in it. The lungs and pericardium were inflamed and in patches were coated

* "Trinamide," May & Baker, Ltd.

with a greyish-red deposit with many inter-pleural adhesions.

*Bacteriology*.—Cultures of the exudate proved to be sterile.

*Histology*.—Typical lesions of feline influenza were present.

**Case 5**

*Subject*.—Four-year-old queen.

This cat had suffered from a persistent cough all winter with occasional periods of inappetence. Severe respiratory disturbance commenced a week before examination. There was pronounced dyspnoea with pain when pressure was applied to the chest, and the animal tended to adopt the prone sternal position already described. Body temperature was 101° F. The left side of the chest produced no respiratory sounds on auscultation and the heart sounds were muffled, this side was also very dull to percussion. On the right side, the respiratory and heart sounds were normal and there was normal resonance to percussion. The heart beats were actually visible on the right side. Destruction was advised.

*Post-mortem*.—The left side of the chest cavity was filled with an offensive purulent creamy fluid containing clots of fibrin. The left lung was collapsed and in places was coated with a greyish deposit. The right side of the thorax was apparently normal.

*Bacteriology*.—A coagulase positive haemolytic staphylococcus and *Bacterium coli* were isolated from the exudate.

*Histology*.—The left lung showed collapse and areas of broncho-pneumonia. There was no evidence of parasites.

**Case 6**

*Subject*.—Ten-year-old neuter cat.

This cat had had what was described as a cold a fortnight earlier, but had apparently recovered and had then developed respiratory difficulty four days previous to the examination. There was severe dyspnoea, and pain was elicited on palpation of the thorax. No respiratory or cardiac sounds were audible on the left side of the thorax which was very dull to percussion. The heart beats were visible on the right side which was apparently normal to percussion and auscultation. The cat adopted the prone sternal position with head held low over the extended forepaws. Body temperature was 100.5° F. Euthanasia was advised.

*Post-mortem*.—The left side of the thorax was filled with an offensive reddish-brown fluid containing clots of yellow material adhering to the pleura of the chest wall and lung. The left lung was collapsed. The right side of the chest was apparently fairly normal.

*Bacteriology*.—A few staphylococci were isolated from the exudate.

*Histology*.—The left lung showed areas of collapse with much thickening of the alveolar and arterial walls. No parasites were visible.

**Discussion**

The cases described above show a striking similarity in the clinical and *post-mortem* picture, but there are obvious differences in the bacteriological and histological findings.

Clinically we have the picture of an exudative pleurisy with pressure collapse of the lungs. The animal adopts the prone position on its sternum with head and neck extended, as this allows the dorsal portion of the lungs to keep above the level of the exudate and thus to perform their function in part. Any pressure on the thorax greatly increases the respiratory embarrassment and also seems to cause pain. Lung and heart sounds are muffled by the blanketing effect of the exudate, and the heart may be displaced by fluid pressure to the right side of the chest. The presence of the fluid also imparts a distinct dullness to the thorax on percussion. The temperature is seldom raised more than a degree, and the pulse is usually quick and thready.

The *post-mortem* picture is very similar in each case with a copious amount of exudate filling one or both halves of the chest cavity. The exudate is usually very offensive in odour and more commonly reddish brown in colour, although when pyogenic organisms are present it was more creamy in character, and contains shreds of fibrin floating in it. The lungs are collapsed and coated with a greyish fibrinous deposit which also extends on to the pericardium. There are frequently adhesions between the pleural surfaces.

Bacteriologically, in half the cases described, the pleural exudate proved to be sterile, despite the fact that it looked and smelt rather purulent. One of the other cases produced a haemolytic streptococcus and another a haemolytic staphylococcus and *Bact. coli*. In the final case it seems probable that the few *staphylococci* isolated were contaminants introduced when the sample was obtained.

The greatest differences occur histologically. Two of the cases showed a heavy infestation with the larvae of *Aleurostrongylus abstrusus* with associated lung damage. Two cases showed lesions of broncho-pneumonia which could be ascribed to a bacterial infection; one case showed the lesions of feline influenza, but the other showed only collapse and thickening of the alveolar and arterial walls, possibly resulting from a past infection.

The explanation as to why such a variety of pulmonary lesions should give rise to similar pleural lesions, must lie in the reaction of the feline pleura to irritation and damage, whatever the aetiology. This reaction consists of the outpouring of the exudate already described and its subsequent effects. It is interesting to note that the irritant agent may be helminthic, bacterial, or viral in nature, but the reaction is almost identical in each case.

**Summary**

1. Six cases of exudative pleurisy in the cat are described.

2. The bacteriology and histology of the cases are recorded.

3. An attempt is made to reconcile the differing pulmonary lesions with the similar pleural lesions.

*Acknowledgment*.—The writer's thanks are due to A. R. Jennings, M.A., M.V.SC., M.R.C.V.S., of the University of Cambridge, for the bacteriological and histological work involved in this study.

# Congenital Atresia of the Large Intestine in a Calf: Successful Surgical Correction

BY

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CONGENITAL atresia of part of the alimentary canal at various levels, though not common, is recognised in babies. Although an extensive search of the literature cannot be claimed, the condition has rarely been mentioned in young animals apart from congenital atresia of the rectum and anus which appears fairly frequently in many species.

During the six months from November, 1954, to May, 1955, three cases of congenital atresia of the distal colon were encountered by us in three unrelated Aberdeen Angus calves. Exploratory laparotomy was carried out in two cases but on finding that the blind end of the colon had become ulcerated, giving rise to extensive acute peritonitis, the animals were destroyed. The third case was of a less advanced nature, the colon being distended but healthy, and it made a good recovery after an anastomosis of the colon to the rectum had been performed.

### *Post-mortem* Findings

The *post-mortem* findings in both cases were essentially similar in character. Both calves were 10 days of age and had been "normal," according to the owners, up till seven days of age. Extensive acute peritonitis was present originating from multiple perforating ulcers of the distal colon. The whole of the colon and the rectum were markedly distended with gas and faecal material, but appeared anatomically normal except that the colon terminated bluntly as it approached the rectum. In each case, starting as a narrow band, the rectum was present with a lumen, but it was apparently normal throughout the greater part of its length. It contained no faecal material. The anus was normal in both cases. The cause of illness in these calves was obviously obstruction of the colon due to congenital atresia.

### History and Clinical Examination of the Third Case

The calf was first seen when four days of age, the owner's only complaint being that he had not seen any faeces passed by the calf. At this time the calf was apparently normal on clinical examination; it was keen to suckle. A dose of liquid paraffin was administered. The calf was seen again on the farm some 30 hours later; no faeces had been passed, and it appeared more dull and less inclined to suckle. Borborygmi were faint and infrequent. Pulse and temperature were within the normal limits. In the light of the *post-mortem* findings already mentioned, atresia of the intestine was suspected and the calf was brought to the surgery the next day with a view to performing a laparotomy. The operation was performed when the calf was six days old and the following notes were made of a clinical examination immediately beforehand:—

The calf was markedly dull, and the abdomen was noticeably distended and slightly tense. Percussion of the abdomen indicated the presence of gas; there was no indication of free fluid in the abdomen. A half-inch rubber tube could be passed through the anus to a distance of some 10 inches where it stopped suddenly. On removing the tube it was quite clean and uncontaminated by faeces. On auscultation of the abdomen borborygmi were completely absent. No other abnormalities could be detected.

### Anaesthesia and Operation

An extensive area of the right flank was clipped and shaved and the calf anaesthetised by inhalation of ether. This anaesthetic proved successful though difficult to control. The usual aseptic precautions were taken and the rubber tube was held in place in the rectum. On entering the peritoneal cavity the distended end of the large intestine was easily found. The rectum, though much less mobile because of the normal mesorectum, was easily identified due to the tube in the lumen. A large amount of gas was released from the large intestine by puncturing the blunt end of the colon with a hypodermic needle. The blunt end was opened and anastomosed to the rectum as far anteriorly as possible—about 9 inches from the anus and 3 inches from the pelvic reflection of the peritoneum. A double row of continuous No. 2/0 chromic catgut sutures were used; the peritoneum and muscle layers were closed with continuous chromic No. 2 catgut sutures, and the skin was closed with Halstead nylon sutures. Intramammary penicillin was smeared liberally on the anastomosis and applied to the wound before closing.

The calf showed a rapid recovery from the anaesthetic, and rose to its feet some 15 minutes after completion of the operation.

### Post-operative Treatment

An hour later exaggerated borborygmi could be heard even when standing a few feet from the calf, and fluid faeces were passed about three hours later; by this time the distension of the abdomen had subsided.

Post-operative treatment consisted of 200 c.c. of dextran* given intravenously immediately after the operation and 1 gramme streptomycin given daily for five days. Three days later the calf developed pneu-

* "Intradex"; Glaxo Laboratories, Ltd., Greenford, Middlesex.

# "Circling Disease" in Sheep in Shetland due to *Listeria monocytogenes*

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STUART YOUNG

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*LISTERIA monocytogenes* infections of farm animals have been described from many parts of the world and in several different species. A comprehensive review of the literature is given by Gray, Stafseth & Thorp (1951).

In the British Isles, *Listeria* infections have been recorded in sheep in association with abortion by Paterson (1940) and in a calf by Harbour (1941). The recovery of the organism from sheep has occasionally been mentioned in the annual reports of veterinary investigation laboratories. No published record can be found of the recovery of the organism from naturally occurring cases of ovine encephalitis in Britain. The name "Circling Disease" was given to an infectious ovine encephalitis caused by *L. monocytogenes* and originally described by Gill (1931) in New Zealand.

The purpose of this report is to record the isolation of *L. monocytogenes* from a naturally occurring encephalitis of sheep in the Shetland Isles in which the clinical symptoms resembled those of "Circling Disease."

* The author is now working at the Montana State College, Agricultural Experiment Station, Bozeman, Montana, U.S.A.

## Clinical History and Symptoms

*Farm 1.*—This flock consisted of 50 sheep comprising ewes and lambs housed continuously from early in January to March 24th, 1955. This flock was put out during the daytime only on March 24th, 25th and 26th. Symptoms were first seen on March 27th and in the following 10 days, four cases occurred, all in 10-month-old lambs. Of these four lambs, one died within 24 hours of showing symptoms; a second was killed in the terminal stages of disease, 48 hours after symptoms appeared (Brain A); a third lamb received daily treatment with chloramphenicol intramuscularly for eight days at 0.5 gramme a day from the sixth day after symptoms appeared until the 15th day after onset when it was killed since there had been very little response to treatment (Brain B); the fourth lamb was treated on four successive days with chloramphenicol intramuscularly at 0.5 gramme a day from the sixth day after onset of symptoms with apparent complete recovery by the 10th day. This last lamb appeared to be normal three weeks after the disease first appeared in the flock.

The symptoms seen in each case commenced with a restless circling movement of the affected sheep,

monia but this resolved gradually during the following five days. No diet restrictions were imposed, the animal being taken to the farm the day after the operation and allowed to suckle. The nylon sutures were removed 10 days after the operation.

The calf, now six months of age, appears to have grown normally.

## Discussion

Louw & Barnard (1955) have succeeded in producing atresia of the intestine in the dog, identical to that found in naturally occurring human cases, by ligaturing the mesenteric blood vessels supplying a short length of the intestine while the puppy was still "*in utero*"; in so doing they have provided very strong evidence to suggest that the cause of some cases of congenital intestinal atresia is interference with the mesenteric blood supply in the foetus. They point out that the terminal blind end of the gut in such cases, though supplied with sufficient blood to maintain viability, may undergo necrosis when the pressure is increased in normal peristalsis, and suggest that a portion adjacent to the atresia should be resected in the treatment of intestinal atresia. Louw and Barnard quote an instance (Macnab, 1955) where such operative procedure resulted in a reduction in the mortality rate from 69 to 33 per cent. Due to our lack of knowledge of these facts at the time, no part of the intestine was resected.

We are, of course, at a loss to know the incidence of congenital intestinal atresia in calves or in the young of other species, and the fact that we encountered three cases in six months in a three-man large-animal practice may well be nothing more than a coincidence.

## Summary

(1) Mention is made of *post-mortem* examinations in two calves with congenital atresia of the distal colon.

(2) A third case is described in which the condition was diagnosed and successfully treated surgically.

(3) A recent report on the origin of intestinal atresia is summarised.

*Acknowledgments.*—We wish to express our thanks to our principal, Mr. W. J. B. Robson, for his constant interest and his permission to publish this report.

## References

Dollar's Veterinary Surgery. (1950). Edited by J. J. O'Connor. Baillière, Tindall & Cox, London.

LOUW, J. H., BARNARD, C. N. (1955). *Lancet.* **II.** 1065.

MACNAB, G. H. (1955). *Proc. R. Soc. Med.* **48.** 303.

Muir's Textbook of Pathology. (1951). Revised by Cappell. Arnold, London.

generally accompanied by the drooping of one ear and an unnatural leg action. One lamb, after circling, tended to stand with neck extended and head tilted upwards with its chin resting against a wall. These signs were evident only for the first one or two days at the most, and were followed by exhaustion and recumbency. Newly recumbent lambs could stand if set up but were powerless to move. Later, they could not stand even when assisted. The appetite was lost early in the course of the disease. Body temperatures on the second day after the onset of symptoms appeared were normal in the three lambs surviving for this period.

Apart from these four cases in lambs, no other case appeared in this flock. Though close contact existed between sheep and cattle on the premises, no cases have occurred in cattle.

The source of infection in this outbreak has not been discovered. Poultry are kept but losses amongst the birds have been few and those that have occurred have been ascribed to leucosis. The premises are free of rodent vermin.

*Farm II.*—Clinical cases resembling "circling disease" occurred in six sheep on a second farm in Shetland. Unfortunately, no opportunity was presented to examine material from these premises so that bacteriological confirmation is lacking. On this second farm, one ewe and two 10-month-old lambs became ill suddenly on March 26th, 1955. The circling movement and drooping ear were the first signs observed. Exhaustion and recumbency followed. Body temperatures remained normal. One lamb died two to three days later but no *post-mortem* examination was carried out. The ewe and the other lamb gradually recovered. Three further cases in lambs occurred within the next two weeks and these all recovered.

### Bacteriological Examination

The head of the lamb killed on Farm I 48 hours after the development of symptoms was forwarded in wet ice by air to the laboratory in Aberdeen (Brain A). On arrival, the cranial cavity was opened and the brain carefully removed.

Portions of cerebrum, cerebellum and medulla were separately ground using a sterile pestle and mortar and emulsified in a small volume of sterile glucose broth. Material from each of the three emulsions was sown on to 10 per cent. ox-blood agar plates and 2 per cent. tryptose agar slants. The emulsions were then stored in the refrigerator at 4° C., and cultures sown from them after one, two, four, and eight days refrigerator storage. Incubation of cultures was at 37° C. under aerobic conditions.

Of the cultures made before refrigeration of the emulsions, those from cerebrum and cerebellum were sterile after 48 hours' incubation. Those from the medulla yielded numerous *Listeria* colonies in pure culture within 24 hours of incubation. Of the cultures made after various periods of refrigeration, those from the cerebrum remained sterile on all occasions. From both the cerebellum and medulla after refrigeration, cultures yielded numerous *Listeria* colonies in pure cultures. In each case the organism was recovered in cultures made after only one day's refrigeration but, as the period of refrigeration increased, cultures yielded an increasing number of colonies of the organism from the same amount of inoculum.

The refrigeration technique was originally suggested by Gray *et al.* (Gray, M. L.; Stafseth, H. J.; Thorp, Frank, Jnr.; Sholl, L. B.; Riley, W. F., 1948) and its value has been confirmed by numerous other workers, *e.g.* Schaef, A. V. I.; de Jong, J. J., and de Jong, J. M. (1951), and Zink, A.; de Mello, G. C.; Burkhardt, R. L. (1951).

Morphologically, the organism in young cultures was a gram-positive cocco-bacillus showing a tendency to bipolar staining. Occasional rod-shaped forms were observed but filaments were not seen. The organism showed sluggish motility after six hours' incubation at 22° C. in glucose broth.

*Cultural Characteristics*

Colonies of the organism on blood-agar after 24 hours' incubation were smooth, white, semi-translucent, uniformly convex and with an entire edge. They were approximately 0.8 mm. in diameter and produced a narrow zone of $\beta$ haemolysis. With further incubation of the culture the colonies increased in size up to 1.2 mm. in diameter and developed a "poached-egg" appearance by 72 hours.

On MacConkey's agar, growth of the organism occurred less readily than on blood-agar and the colonies were smaller in size. In glucose broth, trypsin broth and peptone water, the organism grew and produced a moderate turbidity in 24 hours at 37° C. With incubation of cultures in these media beyond three days a fine granular deposit developed though turbidity remained moderate. Fermentation studies on the organism in carbohydrate media showed that acid without gas was produced in glucose, sucrose, maltose, salacin, inulin, rhamnose, trehalose, dextrin and mannose. No acid was produced in lactose, mannitol, raffinose, arabinose, xylose, dulcitol, glycerol, galactose, or sorbitol. Methyl red and Vosges-Proskauer tests were negative; neither indole nor $H_2S$ were produced; gelatin was not liquefied. Colonies of the organism of agar slants showed catalase activity.

*Pathogenicity*

Mice inoculated by the intraperitoneal route with 0.5 c.c. of 24-hour broth cultures died within 48 hours and the organism was recovered in cultures from the liver, spleen, kidney, and brain.

A rabbit was inoculated intravenously with 0.5 c.c. of a suspension of the organisms in saline, standardised to Brown's tube 6. The rabbit became dull three days later and remained so until it died five days after inoculation. A blood-cell count on a sample of blood from the rabbit at the time of inoculation showed the monocyte count to be 1,500 per cu. mm. A blood sample taken prior to death on the fifth day had a monocyte count of 5,820 per cu. mm. This represented an increase in circulating monocytes from 15 to 30 per cent. of the total white blood-cell count. At autopsy, the liver of this rabbit was found to be studded with multiple, minute, white foci of

(*Concluded at foot of col. 1, page 461*)

# Current Literature

## REPORT

**Grassland Research Institute. Experiments in Progress, Number 8.** 1956.

This report covers the work of the Grassland Research Institute and is indicative of the intensive effort being given to grassland production in this country. It reflects the world position in grassland production established by the Station, and the vast range of its work. The application of its findings will undoubtedly be reflected in the management and condition of the livestock in the country as they have been in the past; and it would seem that the inclusion of those with veterinary qualifications on the staff, and particularly on the governing body, might well have a considerable contribution to make to future progress. The experiments make very interesting reading.

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**"Circling Disease" in Sheep, etc.**—*concluded.*

necrosis. A bacteriological examination of the rabbit's tissues resulted in the recovery of the organism in cultures made directly from the medulla. Cultures made from a liver emulsion after refrigeration overnight also yielded the organism, though direct cultures from the unrefrigerated liver remained sterile.

The bacteriological examination of Brain B from Farm I did not result in the recovery of *Listeria* organisms. This may have been due to the prolonged treatment the lamb had received prior to its death.

Bacteriological examinations were also carried out on several nasal swabs obtained from these two premises from in-contact lambs and ewes. These examinations did not result in the recovery of *L. monocytogenes*.

### Summary

The clinical history, symptoms, and bacteriological findings in an outbreak of encephalitis of sheep in two flocks in Shetland has been described. This condition was identical to that recognised as "Circling Disease" and *Listeria monocytogenes* was recovered from the brain of one sheep in one of the flocks. The source of infection was not discovered.

*Acknowledgments.*—I would like to thank Mr. David S. Clouston, M.R.C.V.S., for making these cases known to me, and Mr. Ronald Boyne for his technical assistance in the laboratory examinations.

### References

GILL, D. A. (1931). *Vet. J.*, **87.** 60.
GRAY, M. L., STAFSETH, H. J., & THORP, FRANK. *Am. Vet. Med. Assoc.* **118.** 242.
———, ———, ———, SHOLL, L. B., & RILEY, W. F. (1948). *J. Bact.* **55.** 471.
HARBOUR, H. E. (1941). *Vet. J.* **97.** 401.
PATERSON, J. S. (1940). *J. Path. Bact.* **50.** 197.
SCHAEF, A. U. I., DE JONG, J. J., & DE JONG, J. M. (1951). *Tijdschr. voor Diergeneesk.* **76.** 751.
ZINK, A., DE MELLO, G. C., & BURKHARDT, R. L. (1951). *Amer. J. vet. Res.* **12.** 194.

## DIGEST

### Anaemia and Cats

In a review of 120 cases of anaemia in cats, Holzworth (1956), concluded that cats were much more liable to suffer anaemia than other domestic animals. The anaemias were classified into three main groups: haemorrhage due to trauma, haemolytic anaemia due to the destruction of red blood cells, and an interference with erythrocyte production. However, most of the affected cats suffered from a haemolytic anaemia which appeared to be a common complication of a wide range of feline ailments, and frequently associated with localised bacterial infections. The symptoms of inappetence, weakness, pallor, and severe dyspnoea due to anoxaemia, were common to all forms of anaemia. The dyspnoea due to anaemia, is usually associated with the cat lying on its side, in contrast to lung affections, where the cat prefers to adopt the sternal position. An indication as to the type of anaemia was obtained by blood examination, for which the author advises a stained film, and, in his review, he illustrates the main characteristics of the various types of anaemia, with colour plates. Treatment consisted of the use of antibiotics, and the administration of liver extract, iron, and blood transfusions.

### Udder Insufflation and Milk Fever

Fifty years have passed since the Danish veterinary surgeon, Schmidt, cured milk fever in cows, by insufflation of the udder, with air. Although it is generally accepted that milk fever is the result of a low blood calcium, it is recognised that a number of cases respond better to udder insufflation than to intravenous calcium. In an attempt to explain this phenomenon, Mashak (1956) studied the effects of udder insufflation on the blood and colostrum, at intervals immediately following treatment. The conventional method for air insufflation was used, and the teats closed with tapes for two hours. As a result of this treatment, there was a rapid rise in blood calcium, inorganic phosphorus, and sugar, a slight increase in magnesium, but no change in the serum proteins. Mashak suggests that the pressure in the insufflated udder is greater than the secreting pressure of the gland acini with the result that the calcium and other minerals are forced back into the blood-stream. This factor, and the temporary cessation of the secretion of the colostrum, allows the normal mechanisms which adjust blood calcium levels to regain control. The use of udder insufflation is recommended in cases where the response to calcium therapy is poor.

### Fish Noises

The findings of an investigation by the U.S. Office of Naval Research (1956), suggest that the depths of the ocean are far from silent. Underwater animals are as garrulous in giving expression to their emotions as their non-aquatic colleagues. Equipped with hydrophones, American workers have tape-recorded the sounds of many species of marine animals. Fish do not possess vocal chords and communicate by

rattling portions of their anatomy, or by vibrating the walls of their air bladder. In consequence a startling variety of sounds are produced, ranging from a foghorn boom by the toadfish to the pop and crackle of shrimps. Sonic fish become most articulate at night, when spawning, and at the sight of food, but are silenced by the approach of a ship or predator.

D. W. J.

**References**

ANON. (1956). *Nature*, **177**. 1117.
HOLZWORTH, J. (1956). *J.A.V.M.A.* **128**. 471-88.
MASHAK, R. R. (1956). *Ibid.* **128**. 423-30.

## BOOK REVIEW

**Progress in the Physiology of Farm Animals.** Volume II. Edited by J. Hammond. Butterworth's Scientific Publications, London. Price 45s.

This second volume of *Progress in the Physiology of Farm Animals* has four sections covering certain aspects of growth, animal behaviour, locomotion, and reproduction which lead on from the sections of nutrition and environment of the first volume in the series of three.

Half the entire volume is devoted to the section on growth, and of this section about half (more than 110 pages) is concerned with conformation and body composition. The other two sections are concerned with live-weight growth and wool growth. Some aspects of growth are reviewed twice in this section and, indeed, were also considered in Volume I of the series. This section contains a mass of information giving an account of many experiments on carcase production and applied nutrition, many of them bearing the well-known stamp of the Cambridge school of agricultural physiologists. The first two chapters have very extensive lists of references but the reader of the chapter on wool growth is referred to a review prepared by Dr. Marston for Nutrition Abstracts and Reviews.

The second section, on animal behaviour, is concerned with two aspects of the subject, *viz.*, the behaviour of grazing animals and sexual behaviour. Dr. Tribe discusses the validity of the methods used to assess grazing behaviour and shows the interaction of the animal and various environmental factors. Dr. Walton reviews sexual behaviour from the nervous and hormonal point of view as well as the interaction of these two systems.

The section on locomotion by Professor Ottaway is interesting because it gives an account of anatomy from the dynamic point of view.

The section on reproduction contains a single chapter on egg production and fertility in poultry. This appears to be an excellent review of the subject written by Dr. Fraps of the U.S. Department of Agriculture.

This book can be recommended as a whole, but in parts it is difficult reading because of the very great length and detailed accounts of some of the chapters. It is likely that the chapters on behaviour and locomotion will appeal most to veterinary surgeons in agricultural practice. The third volume, not yet published, will probably extend the discussion of reproduction to species other than poultry.

## The University of Liverpool

*As noted in our columns last week, an appeal for half a million pounds towards the University's rebuilding fund has made an auspicious beginning. Part of the fund is to be used for new buildings for the Veterinary School. The article below, describing the history and work of the school, was published in the* Liverpool Daily Post *and is reprinted by courtesy of the Editor, and of the author, Professor J. G. Wright.*

Although veterinary colleges had been established in this country since 1791, when the Royal Veterinary College of London was founded, and others had followed in Edinburgh, Glasgow and Dublin, it was not until 1904 that a university brought veterinary teaching within its academic structure. In that year the University of Liverpool, supported by the Lord Mayor and the Chamber of Commerce, invited Mr. W. Owen Williams, Principal of one of the two veterinary colleges then situated in Edinburgh, to transfer his school to Liverpool where there already existed in the university an Institute of Comparative Pathology.

The prime mover in this project was Sir Rubert Boyce, then Professor of Pathology in the university, and it was largely due to his indefatigable efforts, in which he had the enthusiastic support of a number of prominent veterinary surgeons practising in the city and district, that the school was born. Mr. Williams was appointed Professor of Veterinary Medicine in the university, the school became part of the Faculty of Medicine, and the teaching of the general medical sciences was undertaken by the appropriate departments of the university; a whole-time lecturer in veterinary anatomy was appointed while clinical instruction was undertaken by practising veterinary surgeons who were appointed part-time lecturers.

It was, however, a very puny child and its early life was precarious. It was without endowment and its buildings consisted of a house in Brownlow Street. But despite disappointments and discouragements, the determination and self-sacrifice shown by the handful of men responsible for the school in those days ensured its survival. During the succeeding 15 years progress was slow because of financial stringency; nevertheless, a distinct advance was made by the creation of two Chairs, one in Veterinary Anatomy in 1919, financed from the general funds of the university and the second, the William Prescot Chair in Veterinary Pathology in 1925.

### For the pets of the poor

During the period 1926-1929 four houses in Bedford Street were modified and equipped for teaching and research in veterinary anatomy, pathology and bacteriology. In parasitology there had always been close collaboration with the School of Tropical Medicine, while the city provided facilities for the study of *post-mortem* material and meat inspection at the abattoir.

As yet, however, there was still no provision for clinical instruction and for this the school was dependent on the good offices of veterinary practitioners. In 1928 a veterinary hospital to which Lord Howard de Walden had been a generous subscriber, was built by the university in Brownlow Street and provision made for dealing with horses, dogs and cats. The veterinary hospital did not set out to compete with local veterinary surgeons but limited itself to the free treatment of poor persons' animals and a consultant practice with veterinary surgeons.

One of the greatest difficulties with which veterinary schools, particularly those situated in large cities, have to contend is the provision of an adequate quantity and variety of animals for efficient teaching and research. They have no national service behind them and must find their own. These difficulties will be appreciated when it is recalled that the school's work must relate primarily to the food-producing animals of the farm and in lesser degree to horses and domestic pets. For the latter adequate provision was made by the veterinary hospital, but the school still lacked facilities for the proper training of students in relation to farm animals, and thus they were dependent very largely, as indeed were those of other veterinary schools in this country, on obtaining such practical experience by seeking practice with veterinary surgeons during vacations.

### An outpost at Neston

The imperative need for a maximum agricultural effort during the Second World War focused attention on our veterinary services. As a result a programme of expansion for the Liverpool School was commenced and has been pursued in the years following the war. In 1941, when the Chair of Veterinary Anatomy became vacant, it was decided to attach it to a clinical subject and to link with it the acquisition of a Station in the country where farm animals could be closely studied both in health and disease. A Professor in Veterinary Surgery was appointed and a country house and estate acquired at Neston, Wirral.

Through the good offices of Messrs. J. Bibby and Sons, who placed at the disposal of the School for teaching purposes their two large farms which were situated in the vicinity of the newly acquired Station, a start was made. It was realised that to take full advantage of the facilities provided, and to be available at all times for the study of clinical material, it would be necessary for senior students to live at the Field Station; for this purpose the house Leahurst at Neston was converted into a students' hostel.

Despite war-time difficulties the Field Station was a success from the outset, and since that time it has undergone very considerable extension in land, buildings and staff. It has, in fact, so far entailed a capital expenditure of about £100,000. In 1950, a third Chair was established in the school—Veterinary Preventive Medicine; this department was accommodated at Neston. The Field Station, which now comprises a students' hostel, a farm unit for the teaching of animal husbandry, a hospital for the treatment of farm animals, and laboratory buildings, is still incomplete, and among the further requirements urgently needed are additional student accommodation, animal buildings and increased laboratory facilities. Closely associated with the Field Station there are now seven farms which comprise about 1,000 cattle, some 1,500 pigs, 1,250 sheep and large flocks of poultry which form an invaluable medium for teaching and research in clinical and preventive medicine and obstetrics. There is close consultation with veterinary surgeons practising over a very wide area, and some 150 animals are admitted yearly to the hospital at the Station; the majority are cattle with a sprinkling of horses, pigs and sheep.

We in Liverpool are fortunate in that we have been able to acquire a Field Station which, while being within easy travelling distance of the university and thus allowing close contact with the Departments situated in the city, is close to the intensive farming districts of Cheshire and North Wales. Considerable thought has been given to the division of the student's time. It is accepted that for his adequate instruction he must spend some of his time in the country, yet it is most undesirable that he shall become almost completely detached from the community life and spirit of the university, and every effort has been made to strike a proper balance between the two.

### An expanding department

The Veterinary School of 1956 contrasts strikingly with that of 1904. To-day it comprises a staff of three professors and 22 other full-time veterinary teachers, to say nothing of the close links which exist with the Faculties of Science and of Medicine and with the Tropical School in relation to the teaching of the basic sciences and the fundamental teaching in the subjects of physiology, biochemistry, pharmacology and genetics.

The buildings in Bedford Street have become quite inadequate for the proper housing of the Departments of veterinary anatomy and pathology and among the early future developments of the University is a £250,000 building project for the rehousing of these Departments. The School now has the status of a Faculty, a unique distinction in a United Kingdom university, while under the provisions of the Veterinary Surgeons' Act of 1948, those to whom the degree of B.V.Sc. is awarded are entitled to registration as members of the Royal College of Veterinary Surgeons, the statutory qualification to practise in this country, without additional examination.

The number of students in the School, spread over the five-year course of study, is 150 of which some 10 per cent. are women. In addition there are a number of post-graduate students. The average yearly intake is between 30 and 35. Competition for places is keen and many have to be refused. Provision is made for the annual admission of two or three colonial or other overseas applicants.

# B.V.A. Conference: Supplements and Additives in Animal Feeding-stuffs

**Victoria Halls, Bloomsbury Square, London, W.C.1, on Thursday and Friday, November 22nd and 23rd, 1956**

## PROGRAMME

*Chairman :* Mr. E. R. CALLENDER, O.B.E., M.R.C.V.S.

*Deputy Chairman :* Mr. G. N. GOULD, J.P., M.R.C.V.S.

### FIRST DAY

10.00 a.m. Opening remarks by the Chairman.

### DIETARY SUPPLEMENTS OTHER THAN ANTIBIOTICS

#### Additives for Disease Control in Poultry and Turkeys

10.15 a.m. *Author :* Dr. C. Horton-Smith, B.SC.
10.25 a.m. *Openers :* Dr. S. B. Kendall, B.SC., M.R.C.V.S.
10.30 a.m. Mr. J. E. Wilson, B.SC., M.R.C.V.S., F.R.S.E.
10.35 a.m. DISCUSSION.
11.15 a.m. Coffee.

#### The Use of Hormones and Additives as Growth Promoters in Pigs

11.30 a.m. *Author :* Dr. R. Braude.
11.35 a.m. *Openers :* Professor H. G. Lamont, O.B.E., D.SC., M.R.C.V.S.
11.40 a.m. Mr. R. V. Short, B.SC., M.R.C.V.S.
11.45 a.m. DISCUSSION.
12.45 p.m. Luncheon.

#### Vitamin Supplementation in Animals

2.00 p.m. *Author :* Dr. W. F. J. Cuthbertson.
2.10 p.m. *Openers :* Dr. C. S. Grunsell, M.R.C.V.S.
2.15 p.m. Dr. M. D. Wright, B.A., M.B., B.S.
2.20 p.m. DISCUSSION.

#### Mineral Supplements for Farm Animals

3.00 p.m. *Author :* Dr. J. Duckworth, D.SC., F.R.S.E.
3.10 p.m. *Openers :* Mr. S. L. Hignett, B.SC., M.R.C.V.S.
3.15 p.m. Mr. G. N. Gould, J.P., M.R.C.V.S.
3.20 p.m. DISCUSSION.
4.14 p.m. Summary of the day's discussion by Mr. Wm. C. Miller, M.R.C.V.S., F.R.S.E.

*Closing 5 p.m.*

### SECOND DAY

9.00 a.m. Opening remarks by the Chairman.

### ANTIBIOTICS AS DIETARY SUPPLEMENTS

#### Poultry

9.15 a.m. *Author :* Dr. R. White-Stevens (U.S.A.).
9.35 a.m. *Openers :* Dr. W. P. Blount, F.R.C.V.S., F.R.S.E.
9.40 a.m. Dr. M. E. Coates.
9.45 a.m. DISCUSSION.

#### Pigs

10.25 a.m. *Author :* Mr. I. A. M. Lucas, M.SC.
10.35 a.m. *Openers :* Mr. J. O. L. King, M.V.SC., B.SC., M.R.C.V.S.
10.40 a.m. Dr. W. M. McKay, M.R.C.V.S.
10.45 a.m. DISCUSSION.
11.30 a.m. Coffee.

#### Ruminants, Including Foals and Other Animals

11.45 a.m. *Author :* Dr. J. W. G. Porter, M.A.
11.55 a.m. *Openers :* Dr. S. J. Edwards, F.R.C.V.S.
12.00 noon Dr. K. C. Sellers, B.SC., M.R.C.V.S.
12.05 p.m. Major J. H. Wilkins, B.SC., M.R.C.V.S.
12.10 p.m. DISCUSSION
12.45 p.m. Luncheon.

#### High Level Feeding in Disease Control

2.00 p.m. *Author :* Mr. G. N. Henderson, B.SC., M.R.C.V.S.
2.10 p.m. *Openers :* Mr. J. D. Blaxland, M.R.C.V.S.
2.15 p.m. Dr. D. Luke, B.SC., M.R.C.V.S.
2.20 p.m. DISCUSSION.

#### Antibiotics in Relation to Public Health

2.50 p.m. *Author :* Dr. J. M. Ross (Ministry of Health).
3.00 p.m. *Openers :* Mr. J. N. Ritchie, C.B., B.SC., M.R.C.V.S., D.V.S.M.
3.05 p.m. Professor R. Lovell, D.SC., PH.D., M.R.C.V.S., D.V.S.M.
3.10 p.m. DISCUSSION.

#### Mode of Action of Antibiotics as Dietary Supplements

3.45 p.m. *Author :* Dr. J. H. Taylor, M.R.C.V.S.
3.55 p.m. *Opener :* Dr. S. K. Kon.
4.00 p.m. DISCUSSION.
4.30 p.m. Summary of day's discussion by Dr. W. S. Gordon, C.B.E., M.R.C.V.S., F.R.S.E.

*Closing, 5 p.m.*

# News and Comment

## AN IMPORTANT CONFERENCE

On another page we print the programme of an autumn Conference, convened by the Association, which should prove of particular interest to the profession. Within recent times this journal has published a number of papers on the same general subject, that of supplements and additives in animal feeding-stuffs, explaining their advantages and pointing to their possible limitations. Now a body of experts has been called together to review and to discuss the whole subject, which is of so much importance to veterinarians, and through them to agriculture the world over.

Of secondary, but none the less real, value will be the dispelling, among lay people, of some exaggerations and misconceptions which have tended to force the topic out of perspective. The publication of the Conference Proceedings will add to the literature a timely and almost indispensable volume.

## THE PROFESSOR G. H. WOOLDRIDGE PORTRAIT FUND

The subscription list, which has already been contributed to by 176 members of the profession, will remain open until August 31st to give all those who wish to contribute full opportunity to do so. Intending contributors are asked to note this closing date. Individual subscriptions are limited to a maximum of £1 os. od.

Enquiry has been received as to whether corporate bodies, societies, etc., can subscribe and whether such subscription is limited. The Royal College states that subscriptions will be welcomed from such bodies and there is no limit to the amount in those cases.

It is intended to publish a list of contributors each fortnight in THE VETERINARY RECORD. The second list is set out below and the third will be published in our issue of July 28th. It is hoped to include in the presentation an album of the names of the contributors

Col. A. C. Aggarwala, D. A. D. Aitchison, Esq., A. Baker, Esq., E. Beaumont, Esq., F. Beckett, Esq., Dr. M. F. Benjamin, W. E. Blackwell, Esq., Professor T. J. Bosworth, R. R. Bugg, Esq., E. R Callender, Esq., Mrs. M. J. Case, R. Daubney, Esq., Dr. G. O. Davies, H. W. Dawes, Esq., H. C Driver, Esq., G. Eaton, Esq., Dr. S. J. Edwards, R. C. U. Fisher, Esq., Professor C. Formston, H. S. Gates, Esq., Dr. W. S. Gordon, J. A. Griffiths, Esq., O. V. Gunning, Esq., H. E. Harbour, Esq., A. J. Harries, Esq., J. C. Hartley, Esq., J. W. Haywood, Esq., T. G. G. Herbert, Esq., Brig. A. G. Heveningham, J. H. N. Hobday, Esq., J. W. H. Holmes, Esq., C. S. M. Hopkirk, Esq., Professor D. L. Hughes, Major C. W. Hume, Miss M. G. Jordon, Brig. G. A. Kelly, N. S. King, Esq., W. W. Lang, Esq., Dr. P. L. Le Roux, M. Mahmoodullah, Esq., G. May, Esq., A. M. Miller & L. J. Wakley, Captain T. M. Mitchell, K. G. Morgan, Esq., Miss J. Osborne, Captain R. N. Phillips, J. Pasfield, Esq., A. E. Payne, Esq., G. S. Peyton, Esq., S. E. Piercy, Esq., Dr. N. Plum, F. J. Richmond, Esq., J. N. Ritchie, Esq., Mrs. A. G. Rogers, M. J. H. Rogers, Esq., Dr. K. C. Sellers, G. W. Serth, Esq., G. K. Shaw, Esq., R. B. Sherriff, Esq., Lt.-Col. P. S. Sparling, W. A. S. Stevens, Esq, R. Swaby, Esq., L. R. Thomsett, Esq., J. T. Turney, Esq., Miss O. Uvarov, D. A. W. Walker, Esq., B. O. E. Walpole, Esq., Sir Frank Ware, J. S. Watt, Esq., R. F. West & others, J. B. White, Esq., Major G. Williamson, T. Wilson, Esq., and A. J. Wright, Esq.

## NEW CHARTER FOR R.V.C.

Her Majesty The Queen has been graciously pleased to approve the grant of a new Charter to the Royal Veterinary College. H.R.H. The Duke of Gloucester, K.G., who was President of the College under its former constitution, has honoured the College by becoming its Patron, an office last held by His late Majesty King George VI.

The new Council has appointed Professor Sir Charles Lovatt Evans, LL.D., D.SC., F.R.C.P., F.R.S., to be Chairman, Lt.-Col. Sir Merrik R. Burrell, BT., C.B.E., to be Vice-Chairman, and Major Christopher York, D.L., to be Treasurer.

## CONFERMENT OF HONORARY DEGREES

Immediately prior to the opening of the Royal Show, honorary D.Sc. degrees were conferred by the University of Durham on Sir James Scott-Watson and Dr. John Hammond. In addressing the University the Public Orator referred particularly to Sir James's great work for the N.A.A.S., and to Dr. Hammond's studies at Cambridge and elsewhere in the physiology of farm animals, in which subject he was a world authority.

## UNIVERSITY NEWS

### Bristol

*Appointments*

Mr. A. I. Wright, B.V.SC. (Liverpool), M.R.C.V.S., has been appointed Lecturer in Clinical Parasitology. Mr. P. Radford, B.SC. AGRIC. (Nottingham), has been appointed Assistant Lecturer in Animal Husbandry.

*Conferment of Degrees*

As a result of the alteration of the time of the final examination from September to June, the B.V.Sc. graduates were for the first time able to share with other graduates in the medical and science faculties in the main degree ceremony of the university year.

The Great Hall of the University, still showing the effects of war-time damage, was packed to capacity, and the 24 veterinary graduands formed only a small proportion of the whole. At the conclusion of the conferment of degrees, the Vice-Chancellor, Sir Philip Morris prefaced his remarks to the graduates with a special word of welcome to the President and Registrar of the Royal College of Veterinary Surgeons. In his address he made special mention of the additional responsibility which would be theirs who were entering the medical, dental, and veterinary professions, and concluded with good wishes to all who, though having obtained their degree, would still have to continue a life of study if they were to make the most of their future.

The ceremony of admittance to membership of the Royal College of Veterinary Surgeons which followed was, on this occasion, in the hands of the President, Professor L. P. Pugh. In a short speech of welcome to the profession Professor Pugh reminded the new members that whilst they had an additional responsibility they would also have the help and guidance of the Royal College in professional matters. Concluding on a more personal note he exhorted them both to temper science with humanity and maintain a true humility in the pursuit of greater knowledge.

*Pass List—Final Examination B.V.Sc.*

Allen, Michael Mark; Bennett, Jean Marjorie; Boutflower, John Charles; Boyd, James William; Bradshaw, Philip Reginald; Cobb, Leon Moseley; Copestake, Daphne Beryl; Copestake, Peter; David, John Samuel Edwards; Gale, Frederick John; Goodwin, Peter John; Horne, Anthony Wyatt; Lester, Walter Antony; Llewellyn, Geoffrey Gilbert; Lloyd, Trevor Sidney; Mills, Barry Masterman; Nunneley, David; Payne, Laurence Noel; Poland, Jennifer; Ray, Dipak Kumar; Reed, Hugh Charles Brent; Singleton, Thomas Edmund; Walton, Grahame Stenson, and Watson, Dinah Cochrane.

**Edinburgh**

The Degree of B.Sc. in Veterinary Science was conferred on the following by the University of Edinburgh at a Graduation Ceremonial held on July 6th, 1956. The degrees were conferred by the Chancellor of the University, H.R.H. The Duke of Edinburgh.

James Spence Baird, M.R.C.V.S.; Michael James Camden-Smith, B.SC.(NATAL), M.R.C.V.S.; Noreen Dale Clark, M.R.C.V.S.; Kenneth Coutts, M.R.C.V.S.; Peter Harold Cribb, M.R.C.V.S.; David Edward Easingwood, M.R.C.V.S.; Paul Herbert Marshall Griffiths, M.R.C.V.S.; Michael Harkness, M.R.C.V.S.; John Hugh Jones, M.R.C.V.S.; Peter Hubert Lamont, M.R.C.V.S.; John Alexander Lawrence, M.R.C.V.S.; Iain Douglas Cameron Muil Leitch, M.R.C.V.S.; John Brian McFerran, M.R.C.V.S.; Thomas Michael McHugh, M.R.C.V.S.; Robert George Matthews, M.R.C.V.S.; James King Miller, M.R.C.V.S.; John Shirley Henry Morrish, M.R.C.V.S.; Gordon Penman, M.R.C.V.S.; David Angus Pratt, M.R.C.V.S.; Alan Corbett Rowland, M.R.C.V.S.; James Rowland, M.R.C.V.S.; John Richard White Sedgwick, M.R.C.V.S.; Richard Mathieson Shires, M.R.C.V.S.; Meyrick Baggott Stephens, M.R.C.V.S.; Michael William Stone, M.R.C.V.S.; Robert Henry Todd, M.R.C.V.S.; David Bertram Tordoff, M.R.C.V.S.; Pamela Charmbury Wastie, M.R.C.V.S.; Michael Mervyn Whitmore, M.R.C.V.S., and George Alexander Wilson, M.R.C.V.S.

## THIRD INTERNATIONAL CONGRESS OF ANIMAL REPRODUCTION

One of the happiest events at the recent successful Congress of Animal Reproduction was the luncheon given on the opening day by the President of the Royal College of Veterinary Surgeons, Professor Pugh, to the Organising Committee of the Congress.

It was a happy thought to enable many of the foreign members of the Committee to see something of traditional College life at the function held in Magdalene College, of which the President is a Fellow.

The guests met in the ancient Combination Room for sherry, and then proceeded to the Hall for lunch. Professor Lagerlöf made a short and charming speech in English on behalf of the foreign and British members of the Committee, thanking the President for his hospitality.

## R.C.V.S. EXAMINATION RESULTS

**Edinburgh**

*Fourth Examination*

Gee, B. D.; Gray, Brian; Hartley, E. G.; Johnson, D. E.; Jones, J. S. L.; Maasri, K. J.; Moore, T. E.; Nyun, M., and Robertson, H. L.

*Final Examination*

Bhasker, C. G.; Brunton, A. S.; Camden-Smith, M. J.; Cameron, D. C. K.; Clark, Miss N. D.; Coutts, Kenneth; Cribb, P. H.; Easingwood, D. E.; Griffiths, P. H. M.; Gyi, K. K.; Harkness, Michael; Hastie, Hamish; Howells, G. J.; Imlah, Peter; Ironside, J. B. R.; Jones, J. H.; Lamont, P. H. (M); Lawrence, J. A. (M); Leitch, J. D. C. M.; Macdonald, D. C. (M); McFerran, J. B.; McHugh, T. M.; Matthews, R. G.; Miller, J. K.; Mohy-Uddin, G.; Morrish, J. S. H. (M); *Penman, Gordon; Pratt, D. A;. Richards, B. G.; Rowland, A. C.; Rowland, James; Sedgwick, J. R. W. (S); Shires, R. M.; Stephens, M. B. (M); Stone, M. W.; Thomas, M. E. R.; Todd, R. H. (S); Tordoff, D. B.; Wastie, Miss P. C.; *Whitmore, M. M.; Wilson, G. A. (M), and Wilson, H. R.

* 2nd class honours.
(M) credit in Medicine.
(S) credit in Surgery.

**Bristol**

*Fourth Examination*

Choy, T. L., and Datt, S. C.

*Final Examination*

Bailur, D. M.

## PERSONAL

**Births**

RILEY.—On June 30th, 1956, to Chippy (*née* Hall) wife of R. Malcolm Riley, B.V.SC., M.R.C.V.S., of Gesford, Wrexham, Denbighs, a daughter, Eleanor Mary, a sister for Anne.

TOWNSEND.—On May 25th, 1956, at Salisbury, Southern Rhodesia, to Jean, wife of Gordon H. Townsend, M.R.C.V.S., Veterinary Officer, Bechuanaland Protectorate, a son, Christopher Peter.

## R.C.V.S. OBITUARY

We record with regret the deaths of the following members of the profession:—

CLARKE, William George, V.I., Ministry of Agriculture, Stormont, Belfast. Graduated Dublin, March 25th, 1915. Died March 15th, 1956.

CORMACK, John Lawrence, Keresley House, Keresley, Nr. Coventry, Warwicks. Graduated Edinburgh December 17th, 1910. Died July 2nd, 1956, aged 69 years.

MCARTHUR, Neil, V.I., Portal Inspection Office, 106, Ann Street, Belfast. Graduated Glasgow July 18th, 1928. Died February 22nd, 1956.

## COMING EVENTS

### July

17th (Tues.). Summer Meeting of the Association of Veterinary Teachers and Research Workers (Northern Region) at The Evans Biological Laboratories, Runcorn, 11 a.m.

18th (Wed.) General Meeting of the Society of Practising Veterinary Surgeons, B.V.A., at the Queen Hotel, Chester, 2.15 p.m.

19th (Thurs.). Annual General Meeting of the Supplementary Veterinary Register Division, B.V.A., at 7, Mansfield Street, Portland Place, London, W.1, 2.30 p.m.

20th (Fri.). Summer Meeting of the Eastern Counties Division, B.V.A., at Cambridge, 11 a.m.

Summer Meeting of the Mid-West Division, B.V.A., at the Berkeley Café, Clifton, 2.30 p.m.

Meeting of the Yorkshire Division, B.V.A., at the Government Buildings, Crosby Road, Northallerton, 2.30 p.m.

28th (Sat.). Annual reunion of the R.V.C. '52 Club at Ye Miller of Mansfield, Goring.

### September

**16th to 22nd (Sun. to Sat.). 74th B.V.A. Annual Congress at Royal Leamington Spa.**

## ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

### Anthrax

*Ches.* Hall Farm, Wimbolds, Trafford, Chester (July 5).

*Flints.* Midnant Farm, Prestatyn (July 5).

*Northants.* Cottesbrooke Home Farm, Cottesbrooke (June 29).

*Northumberland.* Sunilaws Farm, Cornhill-on-Tweed (July 2).

*Renfrews.* Parkhill Farm, Port Glasgow (July 5).

*Salop.* Steele Farm, Steele Lane, Prees, Whitchurch (July 2); Springfield Farm, Ercall Heath, Newport (July 3).

*Westmorland.* Bouthwaite, Selside, Kendal (July 5).

*Yorks.* Haddon Farm, South Crosland, Huddersfield (June 29); Nelson Farm, Alton, Airton, Skipton (July 3); Slade Hooton Hall Farm, Laughton, Sheffield (July 5).

### Atrophic Rhinitis

*Northants.* Kennels Farm, Brigstock, Kettering (July 5).

*Suffolk.* Stanway Green Farm, Worlingworth, Woodbridge (July 3).

### Swine Fever

*Lancs.* Sidbrook Farm, Mawdesley, Ormskirk (July 4); Crook Fold Farm, Charnock Richard, Chorley (July 5).

*Lincs.* Lucas Gate, Leverton; Main Road, Leverton, Boston (July 2); Banks Lane, Heckington, Sleaford (July 3); Church Farm, Cherry Willingham; Church Farm, Wainfleet St. Mary, Skegness (July 4); Outgate, Leverton, Boston (July 5).

*Berks.* Whistley Mill Farm, Hurst, Reading (July 3); The Hut, Victoria Road, Owlsmoor, Crowthorne (July 9).

*Ches.* Grange Farm, Baddington, Nantwich (July 9).

*Devon.* Babvlon, Silverton, Exeter (July 4); Bridge Farm, Stoke Canon, Exeter (July 9).

*Essex.* Copt Hall Farm, Thaxted, Dunmow (July 4).

*Hants.* Jarvis Court Farm, Swanmore, Southampton (July 6).

*Herts.* Piggots Farm, Albury End, Much Hadham (July 9).

*Lancs.* Shaw Head Farm, Lord Lane, Failsworth (July 3); Martin Croft Farm, Haslingden, Rossendale (July 9).

*Norfolk.* Old Hall Farm, Brisley, Dereham (July 5).

*Somerset.* Peace Hay Farm, Sampford Arundel, Wellington (July 3); West View Farm, Ashill, Ilminster (July 4).

*Yorks.* Brickyard Farm, Catwick, Hull (July 4); Bankfield Farm, Barmby on the Marsh, Goole; Fulwood Head Farm, Sheffield, 10 (July 9).

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## CORRESPONDENCE

*The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.*

### Cramp

Sir,—May I refer Miss Joshua to a note over my name in THE VETERINARY RECORD of December 26th, 1942? After 25 years' experience of the racing Greyhound I stand by that article and have seen many Greyhounds relieved of this painful affliction of cramp.

Miss Joshua's statement that " it is well recognised that in the Greyhound the occurrence is irregular even in known affected dogs" is not borne out by fact provided the circumstances remain the same. It we exclude for the moment the cramp which occurs in the Greyhound as a result of unfitness—*i.e.* that which appears when the unfit musculature is asked to follow a lure at a speed and for a distance beyond the present physical powers of the animal—we can concentrate on the fit, well-trained dog which takes cramp and is known as a cramp subject.

No such dog will take cramp if he is taken to a track and raced over the usual middle distance of, say, 525 yards within an hour of his arrival. If, however, he is kennelled for any period over the hour he is liable to contract the complaint and the longer he is kept the more certain he is to succumb. This process may be repeated as often as we like and at any time of the day or evening. No kennelling—no cramp. Kennel the dog for some hours and he takes it without fail.

Pre-racing kennelled greyhounds behave in a variety of ways, but speaking generally, one can divide them into four classes:—

1. Those which go to lie down and do not care whether they race or not.

2. Those who jump up and down behind the closed door nearly all the time they are kennelled.

3. Those which scratch and salivate until a stream of saliva may run from the door and reach a length of 10 ft.

4. Those which lie and fret without visible physical exertion.

The first three classes do not take cramp, ever. Those in Class 1 run to time and vary little in performance. Those in Classes 2 and 3 do not take cramp but vary in their performance according to

the degree of fatigue they have developed and the time they take to recover (which is not necessarily more than a quarter of an hour if exercised). It is in Class 4 that we find our cramp Greyhound and it is this anxiety state which predisposes him. All the time he is shut up, knowing well that he will be racing, but not knowing when, he lies in a state of nervous tension, secretes no saliva, excretes no urine. When taken out of the kennel he looks normal. He may urinate before he races and he walks normally even for a quarter of an hour but he seldom gets longer than that before he is asked to race.

I believe the cause to be either—(1) that the muscular system contains waste products produced as a result of the prolonged nervous tension, lack of exercise and absence of excretion which cannot be eliminated through lack of freedom and time, before he races; or (2) that the extreme tension generated in the dog fatigues the nervous system and again he cannot recover normality in the time.

At any rate he is a subject, and despite the fact that he is kept on the move for 15 minutes, he will drop during his race but very rarely before he has gone 300 yards and usually about 500 yards or so.

Such onsets can be prevented by the simple, regular administration of potassium bromide in medicinal doses—enough to steady his nervous state and not enough to affect his normal (unkennelled) performance.

In the Greyhound world, it would be unusual to use such an animal at stud, and it is remarkable that most cramp subjects in my experience have been males.

The treatment of Scottie cramp is outside my province, yet there are certain similarities between those cases recorded by Miss Joshua (*Vet. Rec.* **68.** 411) and the racing Greyhound. A cramp Greyhound for instance would not take an attack after six 100-yard sprints. That would cure him for the present. I would, however, like to know from Miss Joshua under what conditions affected Scotties are kept and if indeed really adequate exercise is given to allow of free elimination of waste products and toning up of muscles. Is the dog fretting to go out before he goes?

I am interested in Miss Joshua's report of family incidence for I personally believe that the condition arises from the use of a dog at stud whose nervous tendencies, not always noticeable, have been inherited.

Like Miss Joshua's experience in the treatment of the Scottie, I had tried all the treatments she mentions without success—sedation excepted.

In the condition as I meet it, I feel that the proper approach is central rather than terminal.

Yours faithfully,

J. K. BATEMAN.

The Hook,
Northaw,
Nr. Potters Bar,
Middx.

*July 2nd*, 1956.

**Folic Acid Antagonists in the Control of *Eimeria tenella***

Sir,—An earlier report (Joyner & Kendall, 1955), which followed that of Lux (1954), described the synergic effect observed between pyrimethamine and a sulphonamide used for the control of *Eimeria tenella* and made reference to the possibility that other anti-malarial drugs might be able to replace pyrimethamine as synergists of sulphonamides.

Work at this laboratory has confirmed that the ability to potentiate the coccidiostatic effect of a sulphonamide is shared by a number of substances including proguanil (Paludrine) and two related dihydrotriazines and by at least one of the 2 : 4-diaminopteridines. One of the dihydrotriazines, which is well known as the active product to which Paludrine gives rise in the tissues of an animal being treated for malaria, has, under our experimental conditions, produced some potentiation of sulphadimidine when added to the food at a concentration of as little as five parts per million.

The actual mechanism by which these drugs produce their remarkable effects is still not clear, but it must be of significance that all are powerful antagonists of folic acid which, as is known, is a precursor of the nucleic acids essential to the rapid growth of the schizogonous stages of the life-cycle of *E. tenella*.

The practical application of these findings awaits detailed investigation of the optimum concentrations of the drugs to be used and of the possibility that toxic effects may be observed under field conditions.

Our thanks are due to Messrs. Imperial Chemical (Pharmaceuticals) Ltd., for supplies of proguanil and related compounds and to Messrs. Allen & Hanburys Ltd., for a supply of 2 : 4-diamino-6 : 7-di iso propylpteridine.

Yours faithfully,

L. P. JOYNER,
S. B. KENDALL.

Veterinary Laboratory,
Ministry of Agriculture, Fisheries and Food,
New Haw,
Weybridge,
Surrey.

*July 1st*, 1956

**References**

JOYNER, L. P., & KENDALL, S. B. (1955). *Nature*, London. **176.** 975.

LUX, R. E. (1954). *Antibiotics and Chemotherapy*. **IV.** 971.

ADVERTISER'S ANNOUNCEMENT

BURROUGHS WELLCOME & Co. announce the introduction of "Wellcome" brand Ovine Enzootic Abortion Vaccine (Kebbing Vaccine). This egg-adapted adjuvant vaccine, prepared at The Wellcome Research Laboratories, is intended for the prevention of enzootic abortion in gimmers and ewes and is injected subcutaneously in a dose of 1 c.c., before tupping.

"Wellcome" brand Ovine Enzootic Abortion Vaccine (Kebbing Vaccine) is issued in containers of 20 c.c.